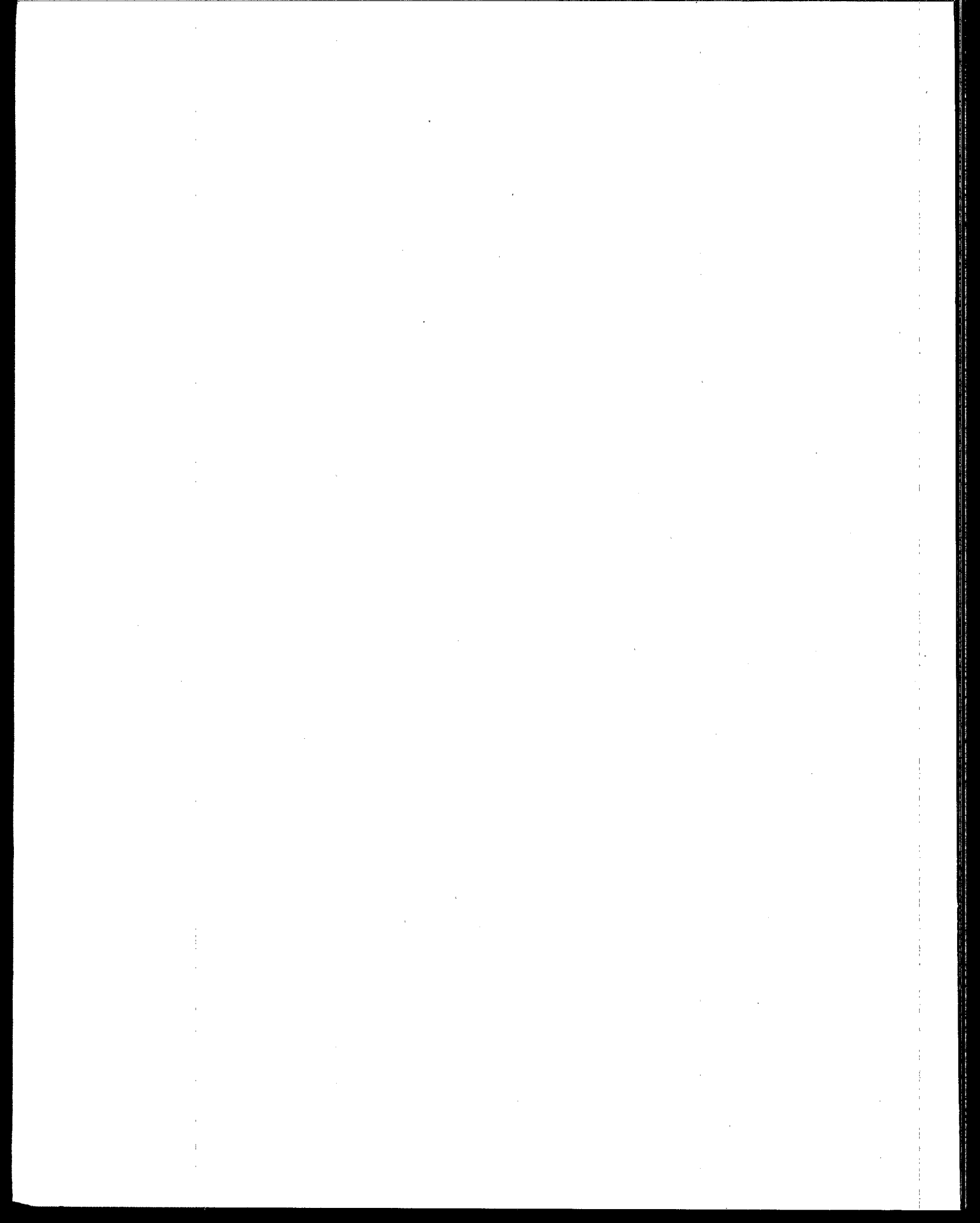




Air Characteristic Study

Volume II

Technical Background Document



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May 1998

Volume II

Air Characteristic Study

Technical Background Document

Office of Solid Waste
U.S. Environmental Protection Agency
Washington, DC 20460



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1.0 Introduction

The U.S. Environmental Protection Agency (EPA) has undertaken a study that evaluates the need for developing a hazardous waste characteristic that addresses risk to human health through the direct inhalation pathway. This air characteristic study includes an analysis of gaps in the hazardous waste characteristics and relevant Clean Air Act (CAA) controls, and the resulting potential risks to human health, posed by the inhalation of gaseous and nongaseous air emissions from wastes managed in certain waste management units (WMUs). These units are: tanks, landfills, wastepiles, land application units, and a subset of surface impoundments. The study covers 105 specific constituents that are volatile organic compounds (VOCs), semivolatile chemicals, and metals.

On November 15, 1996, under a deadline negotiated with the Environmental Defense Fund (EDF), the EPA Office of Solid Waste (OSW) completed the Hazardous Waste Characteristic Scoping Study. This study was conducted to identify potential gaps in the current hazardous waste characteristics, as well as other modifications and updates that are necessary to ensure that the definition of characteristics is complete, up-to-date, and based on state-of-the-art methodologies. Based on the initial bounding analysis of potential risks due to air emissions done as part of the Scoping Study, as well as followup analysis on potential gaps in regulatory coverage under the Clean Air Act and Subpart CC of the Resource Conservation and Recovery Act (RCRA), OSW has identified air emissions from waste management units to be one of the areas meriting further analysis to better characterize potential risks. EPA is under a consent decree to complete the first portion (all units except surface impoundments) of the study by May 15, 1998. The second portion of the study covers surface impoundments receiving wastewaters that never exhibited a characteristic. The surface impoundment study is due to be completed March 26, 2001.

This report describes a national, screening-level analysis designed to assess the potential risk attributable to inhalation exposures when certain chemicals and metals are managed as a waste in certain types of WMUs. Of particular interest are chemicals and metals managed as wastes that are not regulated under RCRA as hazardous wastes. The purpose of this approach is to determine which chemicals and waste management units are of potential national concern, purely from a risk perspective; it is not intended to present conclusions concerning regulatory coverage. This information, combined with preliminary information on regulatory coverage and on the presence of these chemicals in nonhazardous waste, will be useful in determining the need for expanded regulatory coverage. Specifically, the purpose of this study is to provide technical information on potential risk to assist the Agency in determining the need to expand regulatory coverage in the future.

1.1 List of Constituents

The analysis presented in this report addresses specific chemicals that, when managed as a waste, may pose a risk through direct inhalation exposures. Tables 1-1 and 1-2 list the chemicals and metals included in this analysis. Table 1-1 lists chemicals that may be present in nonhazardous industrial waste and that were assessed for all WMUs included in the analysis. Table 1-2 lists chemicals that were included in the analysis of releases from tanks and surface impoundments only.

1.2 Waste Management Units

The waste management units assessed are tanks, landfills, wastepiles, and land treatment units. Because surface impoundments receiving wastewaters that are not hazardous by characteristic are being addressed under a separate study, this analysis does not include surface impoundments as a WMU.

The waste management scenario modeled in this analysis is disposal or treatment of industrial waste streams in RCRA Subtitle D WMUs. For landfills, wastepiles, and land application units (LAUs), the 1985 *Screening Survey of Industrial Subtitle D Establishments* (Shroeder et al., 1987) was the source for data on WMU dimensions and annual waste volumes, along with facility locations for all WMU types. This survey collected information on land-based Industrial D waste management operations in the United States for the 17 industry groups shown in Table 1-3. The resulting data set has been used to represent Industrial D facility locations and WMU characteristics in a variety of RCRA regulatory initiatives, including the Hazardous Waste Identification Rule (HWIR). Although these data are over 10 years old, they represent the largest consistent set of data available.

Tanks were included in this analysis but were not part of the Industrial D survey. Tank characteristics were taken from a previous EPA regulatory analysis supporting the RCRA Subpart CC rules (U.S. EPA, 1991). The tank data used in that analysis were from a 1991 EPA survey of hazardous waste generators and treatment, storage, and disposal facilities (Westat Survey) (U.S. EPA, 1991).

1.3 Organization of Report

The remainder of this report is organized as follows. Section 2 presents a brief overview of the modeling approach. Section 3 discusses waste management unit data and how they were used in the analysis. Section 4 describes the development of emissions for each unit type. Development of dispersion coefficients and health benchmarks are discussed in Sections 5 and 6, respectively. Section 7 describes the development of risk-specific waste concentration distribution. Section 8 discusses how uncertainty and variability are addressed in this analysis. References are provided in Section 9 and supporting analyses are included in Appendixes A through E.

Table 1-1. Constituents Modeled for All WMUs

Constituent	CAS No.
Acetaldehyde [ethanal]	75-07-0
Acetone [2-propanone]	67-64-1
Acetonitrile [methyl cyanide]	75-05-8
Acrolein	107-02-8
Acrylonitrile	107-13-1
Allyl chloride	107-05-1
Arsenic	7440-38-2
Barium	7440-39-3
Benzene	71-43-2
Beryllium	7440-41-7
Bromodichloromethane [dichlorobromomethane]	75-27-4
Bromoform [tribromomethane]	75-25-2
Bromomethane [methyl bromide]	74-83-9
1,3-Butadiene	106-99-0
Cadmium	7440-43-9
Carbon disulfide	75-15-0
Carbon tetrachloride	56-23-5
Chlorobenzene	108-90-7
Chlorodibromomethane [dibromochloromethane]	124-48-1
Chloroform	67-66-3
Chloromethane [methyl chloride]	74-87-3
Chloroprene [2-chloro-1,3-butadiene]	126-99-8
Chromium VI	7440-47-3
Cobalt	7440-48-4
Cumene [isopropyl benzene]	98-82-8
Cyclohexanol	108-93-0
1,2-Dibromo-3-chloropropane	96-12-8
1,2-Dichlorobenzene [o-dichlorobenzene]	95-50-1
1,4-Dichlorobenzene [p-dichlorobenzene]	106-46-7
Dichlorodifluoromethane [CFC-12]	75-71-8
1,2-Dichloroethane [ethylene dichloride]	107-06-2
1,1-Dichloroethylene [vinylidene chloride]	75-35-4
1,2-Dichloropropane [propylene dichloride]	78-87-5
cis-1,3-Dichloropropylene	10061-01-5
trans-1,3-Dichloropropylene	10061-02-6
1,4-Dioxane [1,4-diethyleneoxide]	123-91-1
Epichlorohydrin [1-chloro-2,3-epoxypropane]	106-89-8
1,2-Epoxybutane	106-88-7
2-Ethoxyethanol [ethylene glycol monoethyl ether]	110-80-5
2-Ethoxyethanol acetate [2-EEA]	111-15-9
Ethyl benzene	100-41-4
Ethylene dibromide [1,2-dibromoethane]	106-93-4

(continued)

Table 1-1. (continued)

Constituent	CAS No.
Ethylene oxide	75-21-8
Formaldehyde	50-00-0
Furfural	98-01-1
Hexachloroethane	67-72-1
n-Hexane	110-54-3
Lead	7439-92-1
Manganese	7439-96-5
Mercury	7439-97-6
Methanol	67-56-1
2-Methoxyethanol	109-86-4
2-Methoxyethanol acetate [2-MEA]	110-49-6
Methyl tert-butyl ether	1634-04-4
Methylene chloride [dichloromethane]	75-09-2
Methyl ethyl ketone [2-butanone][MEK]	78-93-3
Methyl isobutyl ketone [hexone] [4-methyl-2-pentanone]	108-10-1
Methyl methacrylate	80-62-6
Naphthalene	91-20-3
Nickel	7440-02-0
2-Nitropropane	79-46-9
N-Nitrosodi-n-butylamine	924-16-3
N-Nitrosodiethylamine	55-18-5
N-Nitrosopyrrolidine	930-55-2
Propylene oxide	75-56-9
Pyridine	110-86-1
Styrene	100-42-5
1,1,1,2-Tetrachloroethane	630-20-6
1,1,2,2-Tetrachloroethane	79-34-5
Tetrachloroethylene [perchloroethylene]	127-18-4
Toluene	108-88-3
1,1,1-Trichloroethane [methyl chloroform]	71-55-6
1,1,2-Trichloroethane [vinyl trichloride]	79-00-5
Trichloroethylene	79-01-6
Trichlorofluoromethane [trichloromonofluoromethane]	75-69-4
1,1,2-Trichloro-1,2,2-trifluoroethane [freon 113]	76-13-1
Triethylamine	121-44-8
Vanadium	7440-62-2
Vinyl acetate	108-05-4
Vinyl chloride	75-01-4
Xylenes, mixed isomers [xylenes, total]	1330-20-7

Table 1-2. Constituents Modeled for Tanks Only

Constituent	CAS No.
Acrylamide	79-06-1
Acrylic acid	79-10-7
Aniline	62-53-3
Benzidine	92-87-5
Benzo(a)pyrene	50-32-8
2-Chlorophenol [<i>o</i> -chlorophenol]	95-57-8
Cresols, total	1319-77-3
7,12-Dimethylbenz[<i>a</i>]anthracene	57-97-6
N,N-Dimethyl formamide	68-12-2
3,4-Dimethylphenol	95-65-8
2,4-Dinitrotoluene	121-14-2
1,2-Diphenylhydrazine	122-66-7
Ethylene glycol	107-21-1
Hexachlorobenzene	118-74-1
Hexachloro-1,3-butadiene [hexachlorobutadiene]	87-68-3
Hexachlorocyclopentadiene	77-47-4
Isophorone	78-59-1
3-Methylcholanthrene	56-49-5
Nitrobenzene	98-95-3
Phenol	108-95-2
Phthalicanhydride	85-44-9
2,3,7,8-TCDD [2,3,7,8-tetrachlorodibenzo- <i>p</i> -dioxin]	1746-01-6
<i>o</i> -Toluidine	95-53-4
1,2,4-Trichlorobenzene	120-82-1

Table 1-3. Number of Industrial D Facilities and 1985 Waste Generation by Waste Management Unit Type

SIC Code	Industry	Landfills				Land Application Units				Wastepiles			
		No. Facilities		1985 Waste (metric tons)		No. Facilities		1985 Waste (metric tons)		No. Facilities		1985 Waste (metric tons)	
2865, 2869	Organic chemicals	17	0.7%	238,575	0.3%	27	0.6%	1,660,572	1.8%	79	1%	43,771	0.06%
3312-3321	Primary iron and steel	202	9%	3,352,467	4%	76	2%	68,932	0.08%	464	9%	5,571,660	8%
2873-2879	Fertilizer and agricultural chemicals	31	1.3%	5,263,323	7%	160	4%	686,728	0.8%	50	0.9%	4,381,910	6%
4911	Electric power generation	155	7%	48,590,046	62%	43	1%	300,637	0.3%	110	2%	755,800	1%
2821	Plastics and resins manufacturing	32	1%	77,539	0%	17	0%	150,518	0.2%	32	0.6%	2,743,691	4%
2812-2819	Inorganic chemicals	120	5%	2,927,324	4%	24	0.6%	97,959	0.1%	98	2%	37,566,042	54%
32	Stone, clay, glass, and concrete	1,257	54%	6,883,106	9%	309	7%	46,099	0.05%	2,528	47%	8,348,981	12%
26	Pulp and paper	259	11%	5,338,781	7%	139	3%	8,128,977	9%	232	4%	1,334,766	2%
3331-3399	Primary nonferrous metals	111	5%	1,250,125	2%	9	0.2%	339,098	0.4%	312	6%	7,966,935	11%
20	Food and kindred products	194	8%	3,268,486	4%	3,128	73%	69,034,641	77%	540	10%	417,940	0.6%
4941	Water treatment	121	5%	143,099	0%	147	3%	8,140,583	9%	48	0.9%	8,238	0.01%
29	Petroleum refining	61	3%	246,952	0%	114	3%	359,888	0.4%	158	3%	71,511	0.1%
30	Rubber and miscellaneous products	77	3%	472,520	1%	16	0.4%	47,448	0.05%	123	2%	53,481	0.1%
37	Transportation equipment	63	3%	155,926	0%	11	0.3%	264	0.0003%	362	7%	644,023	0.9%
2822, 2824, 2851, 2891	Selected chemicals and allied products	21	0.9%	101,953	0.1%	17	0.4%	388,676	0.4%	41	0.8%	6,630	0.01%
22	Textile manufacturing	28	1%	62,655	0.08%	72	2%	693,872	0.8%	103	2%	16,210	0.02%
31	Leather and leather products	9	0.4%	8,460	0.01%	0	0%	0	0%	54	1%	10,201	0.01%
Total		2,757		78,381,336		4,309		90,144,891		5,335		69,941,790	

Source: Screening Survey [Database] of Industrial Subtitle D Establishments (Shroeder et al., 1987).

2.0 Modeling Approach and Data Sources

This section provides a general overview of the modeling approach and primary data sources used and describes how the risk analysis was conducted. Further detail on the models used and a complete set of inputs and associated references are provided in Sections 3 through 8 of this document.

2.1 Overview of Modeling Approach

The overall goal of this risk analysis is to assess the risk to human health via inhalation exposure of 105 constituents managed as waste in landfills, land application units (LAUs), wastepiles, and tanks. The purpose of this analysis was to estimate the concentrations of constituents managed in WMUs that are protective for adults, children, and workers for three different types of exposures or risk endpoints: chronic (1 year), subchronic (1 month), and acute (1 day).

The analytical approach for this analysis was based on three primary components:

- Emissions modeling--characterizing emissions from a WMU
- Dispersion modeling--describing the transport of these emissions through the ambient environment
- Exposure modeling/risk estimation--estimating exposure to a receptor and then backcalculating to arrive at a waste concentration (C_w) that presents a risk equal to a prespecified risk level (e.g., 1 in 1 million or $1E-6$).

The Air Characteristic Study addresses:

- 105 constituents
- 4 WMU types
 - landfill
 - land application unit (LAU)
 - wastepile (WP)
 - tank
- 4 receptors
 - adult resident
 - child resident
 - offsite worker
 - onsite worker
- direct inhalation only
- volatiles and particulates
- 7 distances from the site
- 3 averaging times/risk endpoints
 - chronic (1 year)
 - subchronic (1 month) - LAU, WP
 - acute (1 day) - LAU, WP

Figure 2-1 is a conceptual diagram of a waste site. Constituents managed in the WMU can be released as gases or particles or both. People who live and work at various distances and in various directions from the unit will have different inhalation exposures depending on wind patterns at the site. Workers at the site may also be exposed.

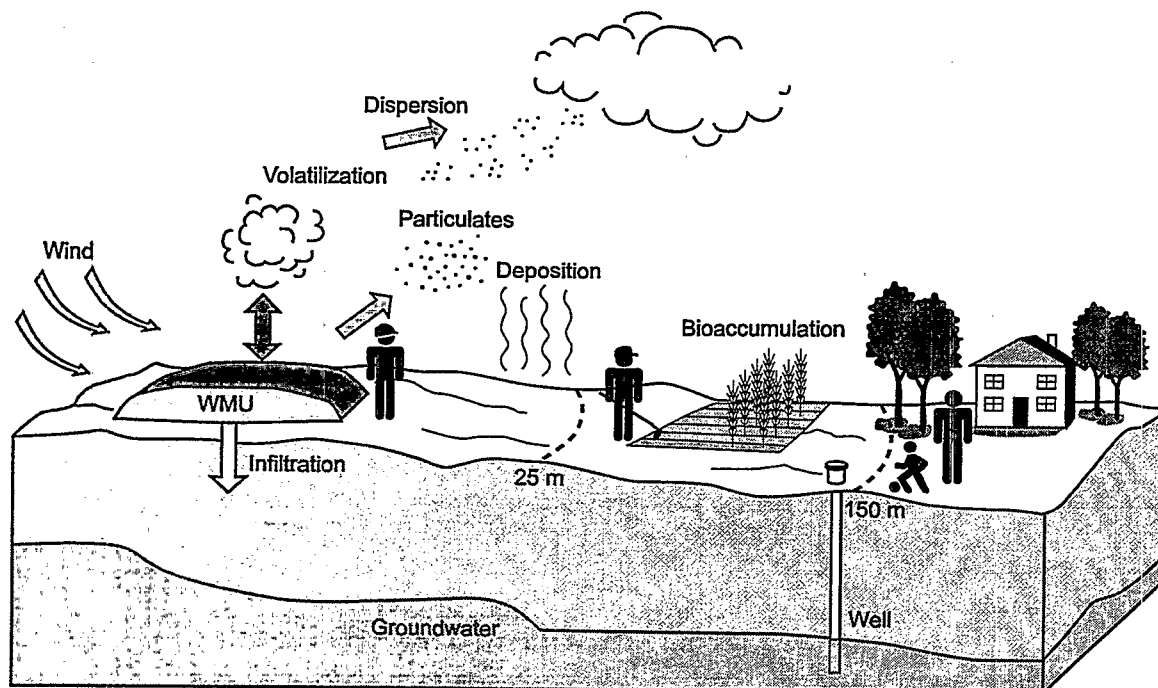


Figure 2-1. Conceptual diagram of a waste site.

Preliminary model requirements for this analysis included:

- Emissions models for the various WMUs to provide estimates of gas and particle releases from the unit
- A dispersion model capable of modeling area sources for chronic (1 year), subchronic (1 month), and acute (1 day) releases
- An exposure model for locating receptors proximate to the WMUs and estimating their exposure
- A risk model that combines the exposure estimate with a dose-response relationship
- The ability to backcalculate C_w from a prespecified risk level (e.g., $1E-6$).

For each constituent and each WMU type, EPA wanted to be able to specify a C_w that would not exceed the risk level (e.g., 1 in 100,000, $1E-5$) in more than a specified percentage (e.g., 10 percent) of the cases being modeled. Therefore, a probabilistic modeling approach, which would produce a distribution of C_w , was needed (rather than a deterministic approach, which would produce a point estimate). A probabilistic approach considers the variability in the inputs required to estimate the concentration nationally. For this analysis, EPA used a Monte Carlo simulation. This is a type of probabilistic analysis in which the distribution

of some or all input variables is known or estimated. A large number of iterations on the calculations are performed (i.e., 1,000), with a value for each input variable selected at random from the variable's distribution and the result (in this case, C_w) calculated for each iteration. The results of each iteration are combined into a distribution of C_w . It was assumed that the modeled cases represent the national distribution of risk-specific concentrations.

To estimate volatile emissions from each type of WMU, EPA's CHEMDAT8 model was used. For the landfill, LAU, and wastepile, the concentration of hazardous constituent in the surface layer of the soil (hereafter referred to as soil concentration) was estimated using a mass balance approach (i.e., competing pathways such as volatilization, adsorption, and biodegradation are accounted for). Particulate emissions due to wind erosion were modeled for land-based units (landfills, LAUs, and wastepiles). Landfills and LAUs were modeled as ground-level sources and wastepiles as elevated sources. To obtain the emission rate of constituent sorbed to particulate matter, the emission rate of particulate matter was multiplied by the soil concentration calculated by CHEMDAT8. Landfills and tanks have a continuous waste loading, while LAUs and wastepiles have noncontinuous, episodic loadings. To capture potential peaks in emissions immediately after loading events, acute and subchronic exposures were evaluated for LAUs and wastepiles.

Dispersion modeling was performed for each WMU using EPA's Industrial Source Complex Model Short-Term (ISCST3) to develop unitized air concentrations (UACs). These are dispersion coefficients based on a unit emission (i.e., $1 \mu\text{g}/\text{m}^2\cdot\text{s}$) for use in a backcalculation. UACs varied depending on the averaging time, the size of the WMU, the distance and direction of the receptor from the WMU, and the associated meteorological station.

The air concentration at any specific receptor is the product of the emission rate (in $\mu\text{g}/\text{m}^2\cdot\text{s}$) and appropriate UAC (in $[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\cdot\text{s}]$). Air concentrations were estimated for chronic, subchronic, and acute exposures, based on a combination of volatile and particulate emissions.

The data used to identify and characterize WMUs contained no information on the location and types of receptors near the facility. Many previous risk analyses have used the maximum point of exposure at some prespecified distance from the WMU as the point for analysis. Such an approach is usually criticized as being overly conservative because it does not consider the possibility of no one living at that exact point. Since individuals may potentially be located in any direction and at various distances from a facility, this analysis developed an explicit way to incorporate this consideration. First, a sensitivity analysis was conducted to determine a reasonable distance at which to bound the analysis. This sensitivity analysis showed that, beyond 1,000 meters, most air concentrations are a small percentage (less than 10 percent) of the concentration at the point of maximum exposure. Therefore, 1,000 meters was used as the outer bound on the distance of receptors included in this analysis. A receptor grid was set up to allow individuals to reside in any of 16 directions and at distances of 25, 50, 75, 150, 500, and 1,000 meters from the edge of the unit.

For this analysis, four receptors were included: an adult resident, a child resident, an offsite worker, and an onsite worker. The onsite worker was located at the 0-m distance or edge

of the WMU. The adult and child resident and offsite worker could be located in any of 16 directions and at distances of 25, 50, 75, 150, 500, and 1,000 m from the edge of the unit. Each distance was evaluated separately and the location of a receptor was allowed to vary among any of the 16 directions. For acute and subchronic exposures, receptors were modeled at 0, 25, 50, and 75 m, because it was assumed that the greatest possibility of acute exposure would be closest to the site.

2.2 Conducting the Analysis

As discussed earlier, the analysis consists of three main parts: emissions modeling, dispersion modeling, and exposure modeling/risk estimation. Figure 2-2 shows the model framework. Emission and dispersion modeling were performed first and the results used as inputs to the exposure modeling/risk estimation (Shroeder et al., 1987). In addition, a database containing characterizations of WMUs was used.

The goal of the analysis is to backcalculate a waste concentration that will result in a specified risk. Because risk is assumed to be linear with waste concentration under most circumstances, this was done by forward calculating a risk associated with a unit waste concentration (i.e., 1 mg/kg for land-based units and 1 mg/L for tanks), then scaling the unit concentration using the ratio of target risk to calculated risk. The assumption of linearity is accurate for the dispersion modeling and the exposure and risk modeling. The emissions model is linear within certain restrictions (e.g., that the concentration does not exceed the saturation concentration). This was accounted for by checking backcalculated results that fell outside the restrictions and modifying any results that did so.

Emissions modeling was performed for all WMUs and all chemicals, assuming a unit concentration of the chemical in the waste (1 mg/kg for land-based units or 1 mg/L for tanks). These emissions were used as inputs to Step 2 of the exposure modeling/risk estimation portion of the model.

Dispersion modeling was performed for 21 representative WMU areas and 29 meteorological locations, assuming a unit emission rate of $1 \mu\text{g}/\text{m}^2\text{-s}$. This produced UACs for each area, meteorological station, and receptor location, which were used as the basis from which to interpolate in Step 4 of the exposure modeling/risk estimation portion of the model.

The analytical framework shown in Figure 2-2 consists of a series of steps and loops. In Step 1, a chemical and WMU type (e.g., landfills) were selected (thus, all landfills were analyzed as a group for each chemical, and so on).

In Step 2, a WMU was selected from the data file for that unit type. For example, for landfills, the database has a data record containing the facility identification and WMU characteristics such as surface area, depth, and waste quantity managed per year for each of 790 landfill units. The database also has a sampling weight for each facility that defines how many facilities were represented by that facility. An assigned meteorological station was added to the database based on locational information for each WMU. The model simulation starts with the

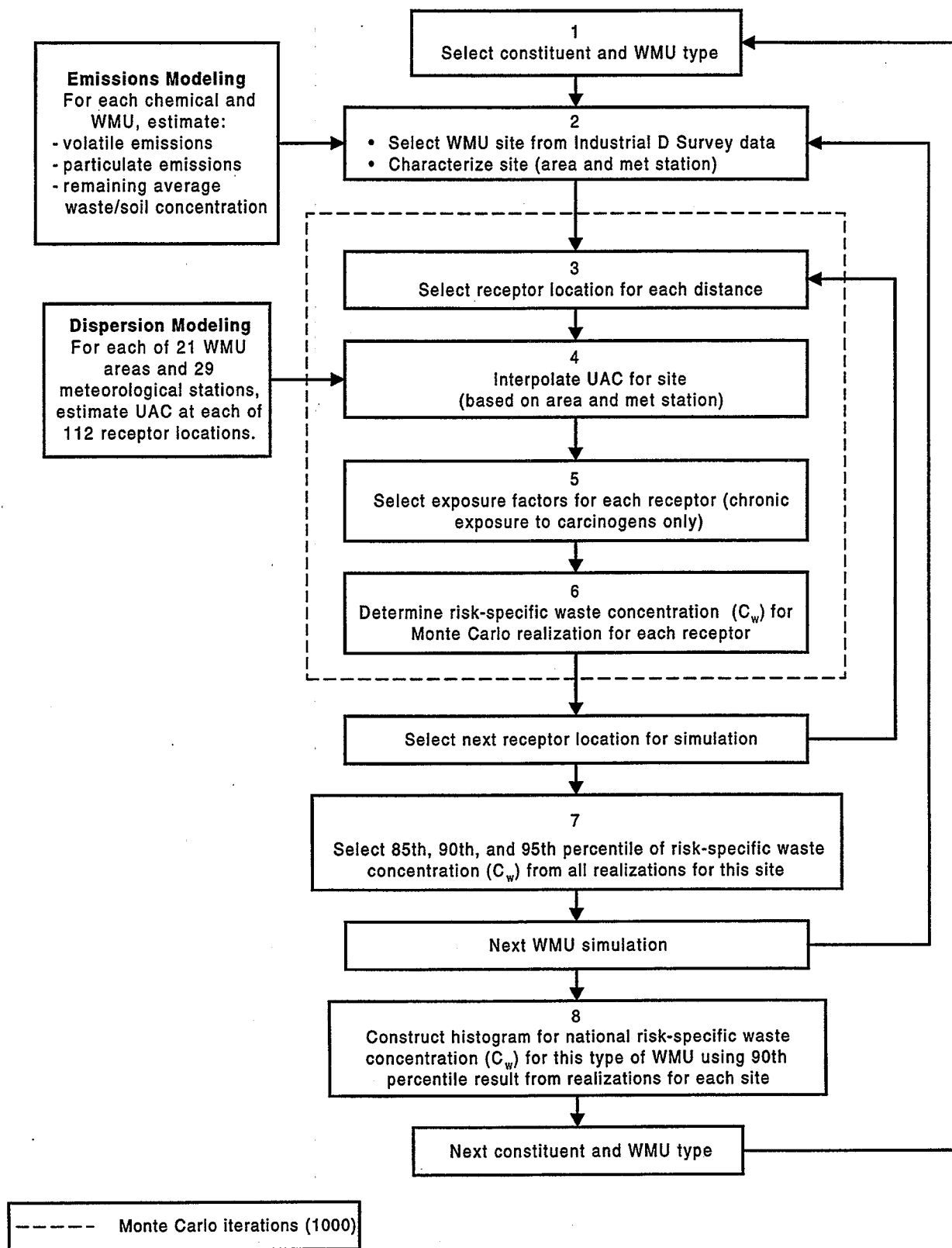


Figure 2-2. Model framework.

first record and moves to each successive record. For each WMU record, the associated emission rate for that WMU and chemical was obtained from the emission modeling results.

In Step 3, receptor locations were selected by choosing at random one of the 16 directions modeled in the dispersion modeling. Receptors were modeled in that direction at each of seven distances from the site.

In Step 4, a UAC was interpolated for the WMU. UACs were modeled for only 21 selected WMU areas for each meteorological station and receptor location. To calculate a UAC corresponding to the WMU's actual area, EPA interpolated between the UACs for the two closest of the areas modeled. For example, the first three areas modeled for wastepiles were 20, 162, and 486 m². For a WMU with an actual area of 100 m², the UAC was interpolated from the UACs for 20 and 162 m². For a WMU with an actual area of 200 m², the UAC was interpolated from the UACs for 162 and 486 m².

In Step 5, for chronic exposures to carcinogens, values of exposure factors such as body weight, inhalation rate, and exposure duration were chosen at random from distributions of these parameters (developed from data in the *Exposure Factors Handbook*, U.S. EPA, 1997) to capture the variability in exposure factors for a given receptor. These exposure factors differ for different receptor types (such as adults, children, and workers). Noncarcinogens were not assessed in this manner because the health benchmarks, such as EPA's reference concentration (RfC), are expressed in terms of ambient concentration and cannot be adjusted for variations in these exposure factors. Similarly, acute and subchronic health benchmarks are expressed as ambient exposure concentrations and cannot be adjusted for variability in exposure factors.

In Step 6, the emission rate, UACs, and, if applicable, the exposure factors, were combined with the health benchmark for the chemical to estimate risk (for chronic exposure to carcinogens) or hazard quotient (for acute and subchronic exposures, and chronic exposures to noncarcinogens) associated with the unit concentration modeled. This risk was then compared to the target risk of either 1 in 1 million, 1 in 100,000, or 1 in 10,000 (i.e., 1E-6, 1E-5, or 1E-4) and the ratio was used to scale the unit concentration to a concentration in the waste (C_w) that would result in the target risk at that receptor. A similar technique was used for scaling the hazard quotient for noncarcinogens.

Steps 3 through 6, which form the core of the Monte Carlo simulation, were then repeated 1,000 times for each WMU, resulting in a distribution of C_w for that WMU for each receptor (adult, child, or worker) at each distance from the site (0, 25, 50, 75, 150, 500, and 1,000 m) for a specific risk criteria (i.e., 1E-4, 1E-5, or 1E-6 for carcinogens and 10, 1, or 0.25 for noncarcinogens). Once 1,000 iterations had been performed for a WMU, various percentiles were selected from the distribution to characterize it. These percentiles represent the percentage of receptors protected at the WMU.

Steps 2 through 6 were then repeated to obtain distributions of C_w for each WMU in the database. These distributions are somewhat different for carcinogens and noncarcinogens and for chronic, subchronic, and acute exposures. For chronic exposure to carcinogens, they represent both the potential variability in location around a WMU, as well as the variability in exposure

duration, inhalation rate, and body weight for each receptor type. For noncarcinogens and for subchronic and acute exposures, variability in these exposure factors is not considered because the measure of risk is a ratio of air concentrations. For chronic exposures to noncarcinogens, the distributions represent the variability in location around the WMU at a specific distance. For subchronic and acute exposures, only point estimates were made at various distances using the receptor located at the point of maximum air concentration for that distance.

The cumulative distribution of C_w for each WMU is presented as the percentage of receptors that are at or below the risk criteria for any C_w (see Figure 2-3, left side). For example, 90 percent of all adult residents at a distance of 150 meters have a predicted risk at or below 1 in 100,000 ($1E-5$) if the concentration of the chemical (e.g., cumene) in the landfill is 1 mg/kg (see point a). A second landfill may have a 90 percent protection level for all adult residents at 150 meters at a concentration of 10 mg/kg (point b), and a third landfill at a concentration of 100 mg/kg (point c). Thus, in Step 7, for each WMU, the distribution shows the percent of potential receptors at or below a specified risk level for each concentration of constituent in the WMU (C_w) for each distance and each receptor.

In Step 8, once all WMUs of a certain type had been modeled, the distributions of C_w for all individual WMUs of the same type (e.g., landfills) were combined to produce a cumulative distribution that presents the variability in C_w across all units of a certain type. For a given percentage of protected receptors (e.g., 90 percent) as described above, the C_w was combined across all WMUs of a specified type (e.g., landfills) to provide a distribution of the percentage of sites considered protective at that level, as shown in Figure 2-3 (right side). Figure 2-3, for example, shows the cumulative distribution of C_w at a 90 percent protection level across all landfills. From this distribution, the 90th percentile C_w value for all 90 percent protection levels across all landfills could be estimated. As described above, three landfills that give a 90 percent protection level (i.e., at $1E-5$) for a resident at 150 meters from the unit boundary have corresponding C_w values of 1 mg/kg, 10 mg/kg, and 100 mg/kg (see points labeled a, b, and c).

These values plus similar values from all other landfills constitute the cumulative distribution. The C_w value that is protective of 90 percent of receptors across 90 percent of the sites is referred to in this study as the 90/90 protection level. These distributions were developed for each unit type, each receptor type, each risk criteria, and each distance from the WMU.

These cumulative distributions are intended to encompass the variability across WMUs. Thus, the variability in WMU characteristics and in meteorological settings are included in these distributions.

This process was repeated from Step 1 for each chemical and WMU type analyzed in this study.

2.3 Data Sources

The Industrial D Survey database (Shroeder et al., 1987) was the primary source of data on WMUs used in this analysis. This database provides information on each of the WMUs assessed, with the exception of tanks. Tank data are from EPA's Background Document

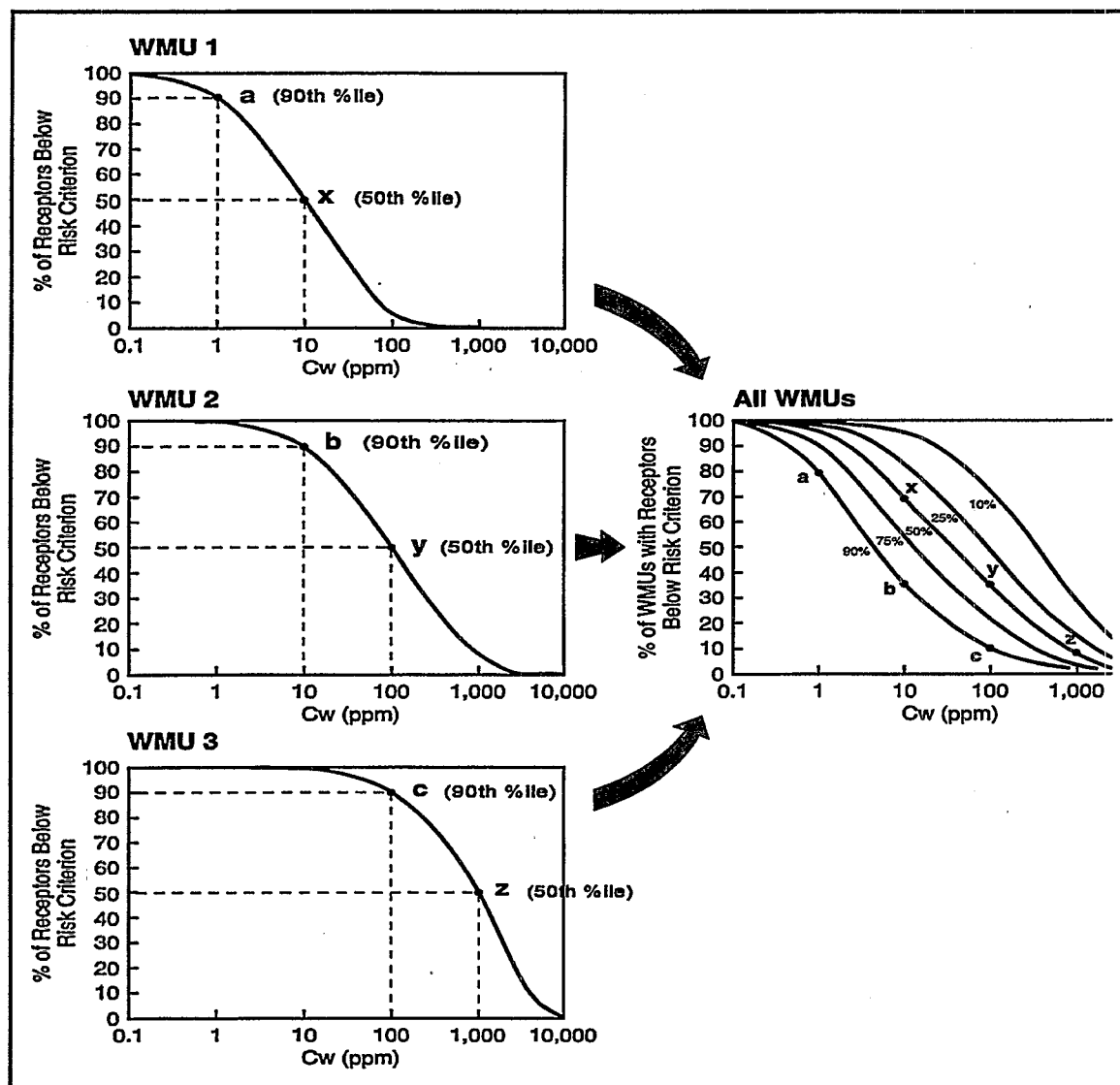


Figure 2-3. Combination of results for individual WMUs into a distribution across all WMUs.

supporting the RCRA Subpart CC rules on air emissions from hazardous waste treatment, storage, and disposal facilities (U.S. EPA, 1991). The Industrial D Survey database contains information on the size and capacity of a statistical sample of each WMU type, general location information, and statistical weights for each facility in the sample. The statistical sample was designed to represent all industrial waste management units not regulated under the RCRA hazardous waste program at the time the survey was conducted in 1987. The weights in the database indicate the number of facilities represented by each facility in the sample. For this assessment, it is assumed that the data contained in this database provide an appropriate representation of the characteristics of each WMU type and of the general location of these types of facilities with respect to climate regions of the country.

Meteorological stations provided temperature and windspeed data as inputs to the emissions model and a large set of inputs for the dispersion model. Although meteorological data are available at over 200 meteorological stations in the United States (see, for example, *Support Center for Regulatory Air Models (SCRAM) Bulletin Board* at <http://www.epa.gov/scram001>), various resource constraints prevented the use of all available data sets in this analysis. Therefore, a set of 29 stations was used that had been selected as representative of the nine general climate regions in the contiguous United States in an assessment for EPA's Superfund Soil Screening Level (SSL) program (EQM, 1993).

In EPA's Superfund study, it was determined that 29 meteorological stations would be a sufficient sample to represent the population of 200 meteorological stations and predict mean dispersion values with a high (95 percent) degree of confidence. The 29 meteorological stations were distributed among the nine climate regions based on meteorological representativeness and variability across each region. Large-scale regional average conditions were used to select the actual stations.

The 29 meteorological stations are listed in Section 5. To assign each Industrial D facility to a meteorological station, EPA used a GIS to construct areas around each station that encompass the areas closest to each station. The boundaries of these areas were then adjusted to ensure that each boundary encloses an area that is most similar in meteorological conditions to those measured at the meteorological station. First, the boundaries were adjusted to correspond to Bailey's ecological divisions (Bailey et al., 1994), which are defined primarily on physiography and climate. The boundaries were further adjusted for coastal (including Great Lakes) areas and the central valley of California to ensure that these stations were used only in regions with similar meteorology. Based on zip codes in the Industrial D Survey database, the Industrial D sites were then overlaid on this GIS coverage and meteorological station assignments were then exported for use in the modeling exercise. Four sites in Alaska and four in Hawaii were deleted from the analysis at this point because the 29 meteorological stations are limited to the continental United States.

3.0 Waste Source Characteristics

Waste sources modeled in the air characteristic risk analysis are landfills, land application units, wastepiles, and aerated and storage tanks. The dimensions and operating characteristics of these units are important determinants of the modeled emission rates and dispersion factors used to estimate inhalation risks to receptors. This section describes how data specific to these unit types were collected and processed for use as emission and air model inputs.

The primary source of data used to characterize waste sources is the Industrial D Screening Survey (Schroeder et al., 1987). These survey data provide information (for all WMU types except tanks) on the number of each type of WMU, the annual quantity of waste managed in the WMU (in 1985), and some information on the dimensions of the WMU (area and capacity). Along with waste concentration, these parameters are the primary waste source characteristics that impact the emission model estimates. The air model also requires WMU area for area sources (landfills, wastepiles, and land application units) as well as the height of the source for wastepiles and tanks. Tank data sources are described in Section 3.4.

Facility locations also were required so that facilities could be assigned to the 29 meteorological stations used to provide meteorological data for the air model. These were derived by matching Industrial D facilities, using the DUNS number, name, street address, and zip code, to EPA's Envirofacts system to obtain the "best" location for each facility as defined by EPA's Locational Data Improvement Project (LDIP) and represented by data in the Envirofacts Locational Reference Table (LRTs) (http://www.epa.gov/enviro/html/lrt/lrt_over.html). For sites that could not be matched to the LRT tables, the latitude/longitude of the facility's zip code centroid was determined and used as the facility location.

3.1 Estimation of Missing Data

The Industrial D Screening Survey included all of the data needed to estimate emissions except landfill depth and wastepile height, which had to be estimated. In the absence of data on wastepile height, two heights, 2 m and 5 m, were chosen. These values, estimated based on the height a frontloader might be able to reach, have been used before in the Hazardous Waste Identification Rule (RTI, 1995). All wastepiles in the Industrial D Survey were modeled twice, once at each height.

Landfill depth was calculated consistent with previous EPA modeling efforts using the Industrial D data (U.S. EPA, 1996a). The following equation was used, assuming the same (fixed by unit type) waste bulk densities used in the previous modeling efforts:

$$\text{depth} = \text{total waste capacity} / (\text{surface area} \times \text{bulk density})$$

The bulk density used for landfills was 1.09577 g/cm³.

Also in accordance with previous EPA modeling efforts using the Industrial D Screening Survey (U.S. EPA, 1996a), landfill capacities were removed from the Industrial D data when depth or capacity constraints were violated. These constraints were imposed to eliminate unrealistic values for depth and capacity. The constraints were: landfill depth had to be greater than or equal to 2 feet and less than or equal to 33 feet, and, within the Industrial D database, landfill total capacity had to be greater than the remaining capacity (U.S. EPA, 1996a, p. 3-6). Of the 790 landfills modeled, 103 had a depth less than 2 feet, 87 had a depth greater than 33 feet, and 21 had remaining capacity that exceeded the total capacity. In addition, 92 facilities were missing data on total capacity, remaining capacity, or both. Thus landfill capacity was missing or screened for 303 landfills.

Capacities to replace the 303 missing or removed values were estimated based on the correlation between surface area and capacity of the waste management unit in the Industrial D data. First, a statistical regression of log (average total capacity) versus log (average surface area) was done on the facilities with known capacities. The regression yielded an equation for a best fit line through the known values. This equation gave the capacity as a function of area, so the missing or screened capacities could be estimated based on the known areas. To provide a more probabilistic sampling of average capacities, and since the known capacities seemed to be in a limited range above and below the best fit line, a positive or negative random number was generated within that range and added to the calculated log (average total capacity) to replace each missing capacity with a random value that was reasonable with respect to landfill area. This value was then used to calculate depth as described above. Figure 3-1 shows the regression plots, including the replaced ("random capacity") values, for landfills.

3.2 Waste Management Unit Data and Size Categories Used in Dispersion Model

The area of a WMU is a very sensitive parameter in the dispersion model. However, because of run-time limitations associated with the air dispersion model, it was necessary to develop area strata for the land-based WMU types. Dispersion modeling was conducted for the median area of each of these strata and the resulting unitized air concentrations (UACs) were used as inputs to an interpolation routine used to determine UACs for each WMU (see Section 5).

Initially, a regular distribution of strata cutpoints (5th, 25th, 50th, 75th, and 95th percentiles) was considered to provide points for interpolation. However, examination of the distribution of areas for each WMU type showed that the distributions were all extremely skewed to the right, with a large number of facilities at the smaller end of the distribution and a few facilities with very large areas. This raised the concern that the regular cutpoints would not adequately cover the tail of the distribution (i.e., WMUs with large areas) for the interpolation process.

To improve the coverage of the cutpoints, the Dalenius-Hodges procedure was used as a starting point. This technique is designed to break the distribution of the known variable X (in

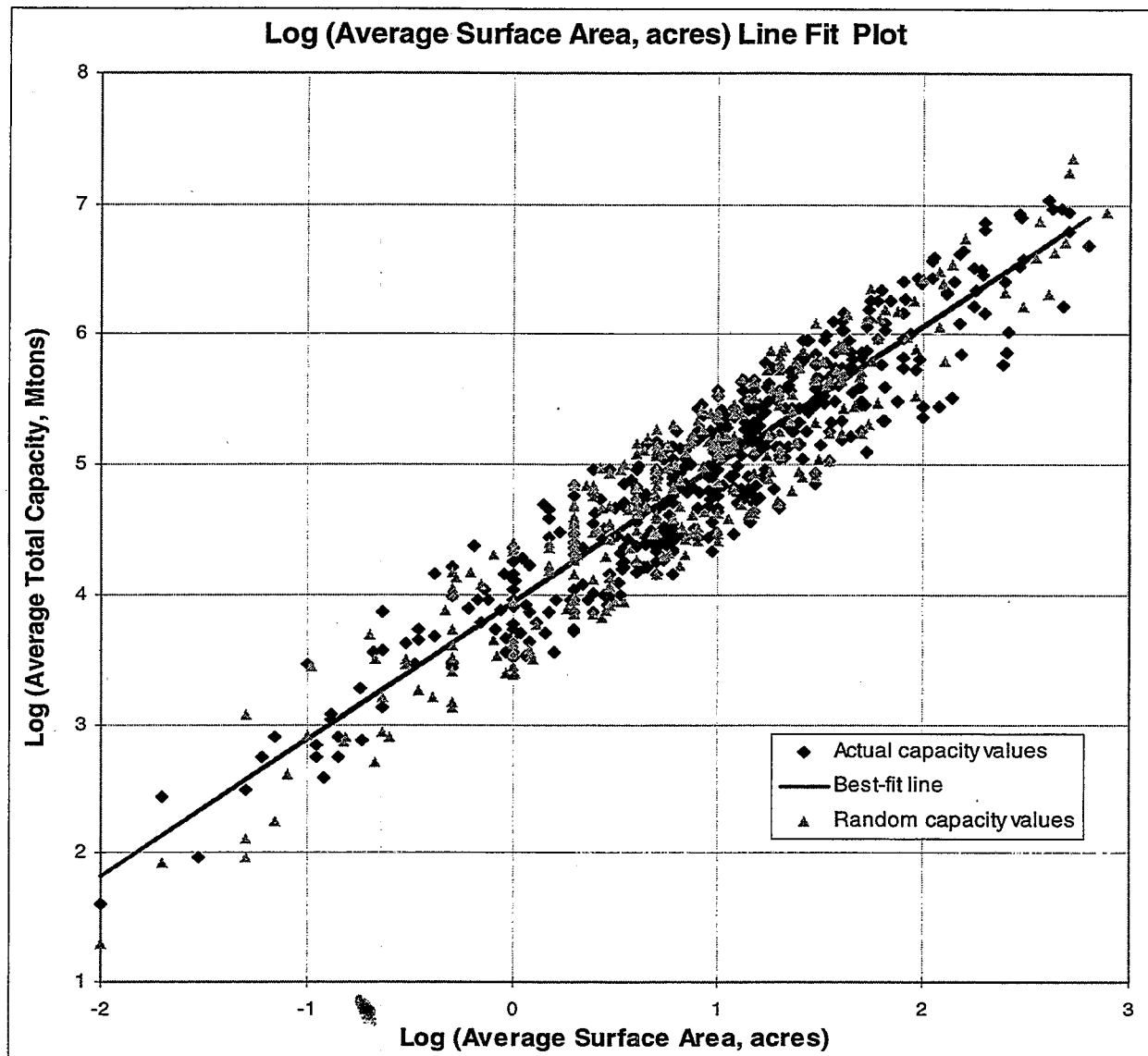


Figure 3-1. Landfill characteristics from Industrial D Screening Survey.

this case, facility area), which is assumed to be highly correlated with the characteristic Y (in this case, the UACs), into L strata in an optimal way. From the L strata, an equal number of observations, n, are picked at random (assuming each observation has an equal weight). (The process was actually employed in this case using the facility weights in the Industrial D data.) To develop the initial WMU strata, L was set at 5; that is, 5 "optimal" strata were initially formed using this approach. This process produces a set of percentiles with the following property: if the distribution is skewed to the right, there will be more emphasis on characterizing the right tail. Appendix A describes the steps of the Dalenius-Hodges procedure in detail.

Based on ISCST3 model run-time estimates, the target number of strata for all WMU types was set at 20. Because land application units and landfills were modeled in the same fashion using ISCST3, it was possible to combine the areas for these WMU types to cover the distribution with as many strata as possible. Fifteen strata were initially formed for this distribution using the Dalenius-Hodges procedure. Wastepiles could not be combined with the other waste management units because they are modeled in ISCST3 as elevated area sources, not as ground-level sources like the others. Five Dalenius-Hodges strata were initially developed for this unit type.

In each case, the Dalenius-Hodges procedure yielded a good characterization of the right tail of the distribution but a poor characterization of the lower portion of the distribution. Because the UACs are most sensitive to area in this region of the distribution, EPA modified the procedure by collapsing two or more of the highest strata into one and successively splitting the lowest stratum into four strata. The splitting was performed by choosing stratum breakpoints as described in Appendix A.

For wastepiles, the Dalenius-Hodges stratification resulted in most (90 percent) of the Industrial D facilities falling into the lowest stratum where more interpolation points are necessary to accurately estimate the UACs. To correct this, the two highest strata (4 and 5) were collapsed into one. The first stratum (1) was then split into two (1a and 1b). This still resulted in a large number of facilities in the lowest strata, so it was split again into two strata (1a' and 1a''). The new lowest strata (1a') was further split into two strata (1a'A and 1a'B). The result was a total of seven strata for wastepiles as shown in Table 3-1.

For the combined landfill and land application distribution, the four highest strata (12, 13, 14, and 15) were collapsed into one and the facility nearest the median of the combined stratum was used to represent that stratum. The next two highest strata (10 and 11) were also collapsed into one and, again, the facility nearest the median of the combined stratum was used to represent that stratum. The first stratum (1) was then split, similar to the wastepiles, into four strata (1A, 1B, 1C, and 1D). The result was a total of 14 strata for the combined waste management units and 21 strata overall.

Table 3-1 shows the final WMU area strata, the medians used for the air dispersion modeling runs and the cumulative percentile of the distribution each stratum represents.

**Table 3-1. Final WMU Area Strata Used for ISCST3 Model Runs
(modified Dalenius- Hodges procedure)**

Strata	Average area (m ²)			Cumulative percentile (Ind D data)
	Low	Median	High	
Wastepiles				
1a'A	5	20	81	48
1a'B	94	162	283	58
1a"	324	486	931	72
1b	1,010	2,100	4,860	90
2	5,200	10,100	44,600	98
3	45,200	101,000	248,000	99
4,5	251,000	1,300,000	2,020,000	100
Combined Landfills and Land Application Units				
1A	14	81	293	17
1B	310	567	789	24
1C	809	1,551	2,293	39
1D	2,307	4,047	7,487	54
2	7,588	12,546	26,980	74
3	27,115	40,500	59,653	81
4	60,300	78,957	119,000	87
5	120,763	161,880	210,000	92
6	210,444	243,000	295,000	94
7	303,525	376,776	546,345	96
8	554,439	607,000	728,460	97
9	753,754	906,528	999,609	98
10 & 11	1,007,703	1,408,356	2,430,000	99
12 to 15	2,521,281	8,090,000	13,500,000	100

3.3 Removal of Wastepiles Containing Bevill-Excluded Wastes

The Resource Conservation and Recovery Act excludes from hazardous waste regulation certain wastes related to mining and mineral processing. These wastes are commonly known as Bevill wastes. Enacted on October 21, 1980, Public Law 96-482 amended RCRA Section 8002 to include subsection (p), which requires the Administrator to study the adverse effects on human health and the environment, if any, of the disposal and utilization of solid waste from the extraction, beneficiation, and processing of ores and minerals, including phosphate rock and

overburden from the mining of uranium ore. Section 7 of these amendments (the Bevill Amendment) amended Section 3001 of RCRA to exclude these wastes from regulation under Subtitle C of RCRA pending completion of the studies called for in Sections 8002(f) and (p). On November 19, 1980, EPA published an interim final amendment to its hazardous waste regulations to reflect the mining waste exclusion (40 CFR 261.4(b)(7)). Since that time, 261.4(b)(7) has been amended to clarify which solid wastes fall under the exclusion as wastes from processing of ores and minerals.

Because an air characteristic would not apply to WMUs containing Bevill Amendment wastes, an analysis of the presence of such facilities in the Industrial D data was conducted to determine whether such facilities would bias the WMU area distributions used in the risk analysis. Identification of facilities that may be handling Bevill wastes was based upon Standard Industrial Classification (SIC) codes assigned to the Bevill waste streams. SIC codes assigned to industries producing Bevill wastes are as follows:

<u>SIC Code</u>	<u>Description</u>	<u>SIC Code</u>	<u>Description</u>
2816	Inorganic Pigments	10 - 1099	Metal Mining
2819	Ind. Inorganic Chem., Nec.	12 - 1299	Coal Mining
2874	Phosphatic Fertilizers	13 - 1399	Oil & Gas Extraction
3312	Blast Furnaces & Steel Mills	14 - 1499	Mining & Quarrying
3331	Primary Copper		of Nonmetallic
3339	Primary Nonferrous Metals, Nec.		Minerals Except Fuels

In addition, electric power facilities (SIC code 4911, electric services) were also included in the analysis because their high-volume waste streams are also exempt from RCRA Subtitle C regulation.

3.3.1 Identification of Bevill Industrial D Facilities

Two approaches were used to identify Bevill facilities from the Industrial D database. The first removed facilities using a relationship between the Bevill-related SIC codes and the industry groups in the Industrial D database:

<u>Industry Group Removed</u>	<u>Group Description</u>	<u>SIC Codes</u>
6	Inorganic Chemicals	2812 - 2819
3	Fertilizer & Agr. Chem.	2873 - 2879
2	Primary Iron & Steel	3312 - 3321
9	Primary Nonferrous Metals	3331 - 3399
4	Electric Power Generation	4911

To provide additional resolution to the analysis (i.e., to reduce the number of facilities inappropriately culled because of the breadth of the Industrial D industry groups), 1,722 Industrial D facilities were matched to EPA IDs and SIC codes in EPA's Facility Index System (FINDS) database. The FINDS SIC codes are multiple per facility and were originally obtained from Dun & Bradstreet. The 1,722 facilities with EPA IDs were then culled by the SIC codes listed above for Bevill and electric power wastes; unlike the "industry group" approach, this

included the mining waste SIC codes. Because of the multiple SIC codes for many facilities, a facility-by-facility review of 4911, 2819, and some mining SIC codes was conducted using RCRIS data to identify facilities that should not be culled (i.e., facilities that could be producing and managing hazardous wastes not covered under Bevill, typically large-quantity generators as indicated in RCRIS). Thus some facilities that would have been culled based on SIC code alone were not removed for the analysis.

3.3.2 Bevill Facilities Culled from Industrial D Data

To decide whether to remove the Bevill WMUs, the area percentiles for each WMU type in the entire Industrial D database were compared with those from the Industrial D datasets with Bevill facilities removed. For landfills and land application units, the area percentiles for the entire Industrial D universe were very similar to those with the Bevill WMUs removed, and the Bevill facilities were not removed for these WMU types. However, removing the Bevill wastepiles resulted in significantly lower wastepile areas for the upper percentile ranges. Based on the SIC codes and facility names, the largest 20 wastepiles could be classified as follows:

- 9 phosphate mines
- 5 other mines
- 6 facilities that do not appear to be mining-related.

Only 3 of the 17 sites with average wastepile area greater than 250,000 square meters were not connected to mining activities.

Based on this assessment, the wastepiles identified as likely to be receiving Bevill waste were identified and removed from the Industrial D WMU data used in this analysis. In total, 82 wastepiles were removed because they contained Bevill wastes, leaving 742 facilities with wastepiles for the risk analysis.

3.4 Tanks

The Industrial D Survey did not include tanks. Therefore, two model tanks were used from the *Hazardous Waste TSDF—Background Information for Proposed RCRA Air Emissions Standards* (U.S. EPA, 1991). These two tanks were located at each of the 29 meteorological locations selected to represent the climate regions of the United States (see Section 5.0), resulting in 58 tanks modeled. Each tank was then modeled four times, using the following assumptions:

- With aeration and biodegradation
- With aeration and no biodegradation
- Without aeration and with biodegradation
- Without aeration or biodegradation.

The tanks modeled under the first two assumptions are referred to as aerated tanks and the tanks modeled under the last two assumptions are referred to as storage tanks throughout this document. Table 3-2 summarizes the characteristics of the two model tanks.

Table 3-2. Input Parameters for Tanks

Input Parameter		Model Plant 1	Model Plant 2
Wind speed (m/s)		Variable	Variable
Temp (°C)		Variable	Variable
Depth/height (m)		4	3.7
Average surface area (m ²)		27	430
Flow rate (m ³ /s)		0.0075	0.088
Active biomass conc. (kg/m ³)		0	0
Biomass in (kg/m ³)	aerated: storage:	0 and 2 0 and 0.05	0 and 2 0 and 0.05
Inlet conc. (g/m ³)		1	1
Inlet COD (g/m ³)		100	100
Total biorate (mg/g-h)		19	19
Fraction agitated	aerated: storage:	0.518519 0	0.581395 0
Submerged air flow (m ³ /s)		0	0
Number of impellers		1	2
O ₂ transfer rate (lb O ₂ /h-hp)		3	3
Power (total, hp)		7.5	120
Power efficiency		0.83	0.83
Impeller diam. (cm)		61	61
Impeller speed (rad/s)		130	130

4.0 Source Emission Estimates

This chapter describes the source-specific emission model and assumptions used to develop the emission estimates for each waste management unit type. Section 4.1 discusses the selection of a general volatilization model and a particulate emission model to use for the emission estimates. Section 4.2 describes some of the critical model input parameters required to run the volatilization and particulate emission models. Subsequent sections describe unit-specific modeling scenarios and assumptions used for the volatilization model effort. The final section of this chapter describes the particulate emission model estimates. Both volatile and particulate emissions were estimated for the landfill, land application unit, and wastepile (referred to as land-based WMUs), while only volatile emissions were estimated for tanks.

4.1 Model Selection

4.1.1 Volatile Emission Model Selection

Several factors were considered in selecting emission models for assessing the potential for contaminant exposure through inhalation. In developing acceptable contaminant limits (exit criteria) for wastes, the ideal emission model would provide as accurate emission estimates as possible without underestimating the contaminant emissions. Because both volatile emissions (for all WMU types) and particulate emissions due to wind erosion (for land-based WMUs) were required in the risk analysis, the volatile emission model had to estimate both volatile emission rates and long-term average soil concentration in the unit (for land-based WMUs). Ideally, the model would provide a relatively consistent modeling approach (in terms of model complexity and conservatism) for each of the different emission sources under consideration. Additionally, the emission model would have to be reviewed both internally by EPA and externally by both State and local agencies and industry representatives. And, the model would have to be publicly available for use in more site-specific evaluations.

Based on these considerations, EPA's CHEMDAT8 model was selected as the model to estimate volatile emissions rates and long-term average soil concentrations in the WMU. The CHEMDAT8 model was originally developed in projects funded by EPA's Office of Research and Development (ORD) and Office of Air Quality Planning and Standards (OAQPS) to support National Emission Standards for Hazardous Air Pollutants (NESHAPs) from sources such as tanks, surface impoundments, landfills, wastepiles, and land application units for a variety of industry categories including chemical manufacturers, pulp and paper manufacturing, and petroleum refining. It also has been used to support the emissions standards for hazardous waste treatment, storage, and disposal facilities (U.S. EPA, 1991) regulated under Subpart CC rules of RCRA, as amended in 1984. The CHEMDAT8 model is publicly available and has undergone extensive review by both EPA and industry representatives. The CHEMDAT8 spreadsheet

model and model documentation may be downloaded at no charge from EPA's web page (<http://www.epa.gov/ttn/chief/software.html>).

The CHEMDAT8 model considers most of the competing removal pathways that might limit air emissions, including adsorption and hydrolysis for tanks, absorption for land-based WMUs, and biodegradation for all units. Adsorption/absorption is the tendency of a chemical or liquid media to attach or bind to the surface or fill the pores of particles in the soil or waste and therefore not volatilize into the air. This tendency to adsorb to or absorb in particles is an important process for estimating the concentration of the chemical on particles emitted to the air due to wind erosion. Biodegradation is the tendency of a chemical to be broken down or decomposed into less-complex chemicals by organisms in the waste or soil. Similarly, hydrolysis is the tendency of a chemical to be broken down or decomposed into less-complex chemicals by reaction with water. Chemicals that decompose due to either biodegradation or hydrolysis have lower potential for emission to the air as gases or particles than those that do not. Loss of contaminant by leaching or runoff is not included in the CHEMDAT8 model. Both leaching and runoff are a function of a chemical's tendency to become soluble in water and follow the flow of water (e.g., due to rainfall) down through the soil to groundwater (leaching) or downhill to surface water (runoff). These two mechanisms would also result in less chemical being available for emission to the air as gases or particles. As such, CHEMDAT8 is considered to provide reasonable to slightly high (environmentally conservative) estimates of air emissions from the various emission sources.

The CHEMDAT8 model was used to estimate the emissions for all WMUs with some minor modifications. For example, for the land-based WMUs (landfills, land application units, and wastepiles), first-order biodegradation rates from the Hazardous Waste Identification Rule (RTI, 1995) were used rather than the biodegradation rate equations in the CHEMDAT8 model. Additionally, certain equations were modified to prevent division by zero when certain volatilization parameters (Henry's law constant or vapor pressure) were zero (e.g., for metals).¹

4.1.2 Particulate Emission Model Selection

The model selection criteria for the particulate emission models were similar to those for the volatilization model. Specifically, the particulate emission model would provide as accurate emission estimates as possible without underestimating the contaminant emissions. The model would provide a relatively consistent modeling approach (in terms of model complexity and conservatism) for each of the different emission sources under consideration, and the emission

¹ Specifically, the CHEMDAT8 model was modified as follows:

- Constituent-specific first-order biodegradation rate constants, applicable for land-based units and converted to units of s^{-1} (see Appendix B).
- If-statements were added to set the biodegradation rate to a negligible level or zero and prevented division by zero when no biodegradation rate constants were available.
- If-statements were added to prevent division by zero for chemicals that did not have vapor pressure, Henry's law constant, or diffusivity inputs (e.g., metals).

model would have to be both reviewed and publicly available for use for more site-specific evaluations. Two different models were selected to model wind erosion: one for wastepiles (elevated sources) and one for landfills and land application units (ground-level sources). Based on the considerations above, the Cowherd model (U.S. EPA 1985b and 1988) was selected for modeling wind erosion emissions from ground-level sources, and the AP-42 model for wind erosion from aggregate storage piles (U.S. EPA, 1985a) was selected for modeling wind erosion emissions from wastepiles. Newer versions of both of these models are available. However, the newer versions are event-based algorithms that require extensive site-specific data that were not available for the sites modeled in this analysis. The versions used probably result in somewhat higher particulate emissions estimates than the event-based algorithms would. This overestimation of particulate emissions is not significant for volatile chemicals, as particulate emissions were found to be a negligible fraction (less than 2 percent in most cases) of total emissions for the volatile chemicals modeled in land-based units. The results for metals other than mercury (which do not volatilize and are therefore based solely on particulate emissions) are somewhat conservative as a result of this overestimation.

4.2 Emission Model Input Parameters

This section discusses the various parameters that impact the estimated volatilization and particulate emission rates. Inputs that influence these rates include input parameters specific to the physical and chemical properties of the constituent being modeled, the physical and chemical characteristics of the waste material being managed, input parameters specific to the process and operating conditions of the WMU being modeled, and meteorological parameters.

A general discussion of the physical and chemical properties of the constituents is provided in the Section 4.2.1. Critical input parameters for the remaining sets of inputs are discussed first for land-based WMU (land treatment) and then for tanks (aerated and storage tanks). A sensitivity analysis was performed to better understand the impact of certain modeling assumptions on the model results. Summaries of the results of the sensitivity analysis are provided in Appendix C for the CHEMDAT8 volatilization emissions model.

4.2.1 Chemical-Specific Input Parameters

Key chemical-specific input parameters include: air-liquid equilibrium partitioning coefficient (vapor pressure or Henry's law constant), liquid-solid equilibrium partitioning coefficient (log octanol-water partition coefficient for organics), biodegradation rate constants, and liquid and air diffusivities. The HWIR chemical properties database (RTI, 1995) was used as the primary data source for the physical and chemical properties for the constituents being modeled. This chemical properties database provided the following chemical-specific input parameters: molecular weight, vapor pressure, Henry's law constant, solubility, liquid and air diffusivities, log octanol-water partition coefficient, and the soil biodegradation rate constants. The CHEMDAT8 chemical properties database (U.S. EPA, 1994b) was used as a secondary data source for the physical and chemical properties for the constituents being modeled. This chemical properties database provided the following chemical-specific input parameters: density, boiling point, Antoine's coefficients (for adjusting vapor pressure to temperature), and biodegradation rate constants for tanks. The biodegradation rate constants in the downloaded CHEMDAT8

database file were compared with the values reported in the summary report that provided the basis for the CHEMDAT8 tank biodegradation rate values (Coburn et al., 1988). Tank biodegradation rates constants for compounds with no data were assigned biodegradation rates equal to the most similar compound in the biodegradation rate database (or set to zero for metals). The specific chemical properties input database used for the emission modeling is provided in Appendix B.

4.2.2 Critical Input Parameters for Land-Based WMU Emission Models

4.2.2.1 Volatile Emissions and Waste Concentration. The input parameters used for the CHEMDAT8 land-based unit emissions model are presented in Table 4-1. (Note: The data entry form in the CHEMDAT8 model refers to oil rather than waste; the term waste is used here for clarity.) Of these parameters, two are actually flags to determine which model equations to apply (Input ID No. L7 and L9). The most important flag for emission estimates is probably the aqueous waste flag (Input ID No. L7). This flag tells the CHEMDAT8 model which equilibrium partitioning model to use between the liquid and gas phases. For organic wastes, the model uses Raoult's law and the liquid-to-air partition coefficient becomes proportional to the contaminant's partial vapor pressure. For aqueous wastes, the model uses Henry's law and the liquid-to-air partition coefficient becomes proportional to the contaminant's Henry's law coefficient. All land-based WMUs were run assuming unit concentration (concentration set to 1 mg/kg, assuming Henry's law applies) and assuming pure component (concentration set to 1E+6 mg/kg, assuming Raoult's law applies).

The annual waste quantity is a critical source (site-specific input) parameter. This parameter along with assumptions regarding the frequency of contaminant addition and the dimensions of the unit combine to influence a number of model input parameters (Input ID Nos. L1, L2, L3, L8, and L12).

The CHEMDAT8 model is insensitive to windspeeds for long-term emission estimates from land-based units. Temperature affects the air diffusivity, which affects the volatilization rate, and potentially affects the biodegradation rate (biodegradation rates were independent of temperature above 5°C and were set to zero below 5°C). Consequently, temperature is the only meteorological data input that potentially impacts the emissions results for the CHEMDAT8 model for the land-based WMU.

The total porosity and air porosity values that were used in the emissions assessments were the default CHEMDAT8 model values for these parameters. These assumed porosity values appear to be reasonable for a waste and waste/soil matrices that have a density of 1.09577 g/cm³.

For aqueous wastes, the molecular weight of the waste (Input ID No. L6) should not impact the calculations. However, CHEMDAT8 converts Henry's law to a mole ratio using the molecular weight of water and then converts back to mass basis using the input waste molecular weight, resulting in an impact in the estimated volatile emission rate (see Appendix C). The CHEMDAT8 model was modified so that the conversion back to a mass basis always uses the molecular weight of water when the aqueous waste flag was set to 1 (alternatively, use 18 g/mol

Table 4-1. CHEMDAT8 Land-Based Unit Model Input Requirements

Input ID No.	Input Parameter	Data Source/Assumption
L1	L, Loading (g waste/cm ³ soil)	Waste quantity and/or density from Ind D Survey
L2	Concentration in waste (ppmw)	1 for unit concentration run; 1E+6 for pure component run
L3	I, Depth of tilling (or unit) (cm)	Assumed or set by capacity
L4	Total porosity	Assumed default value of 0.5
L5	Air porosity (0 if unknown)	Assumed default value of 0.25
L6	MW waste	18 for unit concentration run; 147 for pure component run
L7	For aqueous waste, enter 1	1 for unit concentration run; 0 for pure component run
L8	Time of calc. (days)	Dependent on type of WMU
L9	For biodegradation, enter 1	Dependent on type of WMU
L10	Temperature (°C)	Set by location of WMU
L11	Windspeed (m/s)	Set by location of WMU
L12	Area (m ²)	Input from Ind D Survey

for the molecular weight for aqueous waste). The molecular weight for the "pure component" runs was set to 147 g/mol, which is the CHEMDAT8 default value for this input parameter. If the waste were truly a pure component, then the appropriate molecular weight input is the specific constituent's molecular weight. However, the CHEMDAT8 model does not provide for constituent-specific molecular weights for the waste, and the pure component run is used to backcalculate an appropriate waste concentration limit less than pure component. Therefore, a singular waste molecular weight is appropriate. The molecular weights of the constituents modeled were evaluated. The average molecular weight of the modeled constituents is 120 g/mol; the highest is 272 g/mol. A higher molecular weight increases the estimated volatilization emission rates. As such, the assumed molecular weight value of 147 g/mol provides a reasonable to conservative emission estimate on average for pure components, but may underestimate the volatile emissions for the heaviest compound by a factor of approximately 30 percent.

As biodegradation both lowers the emission rate and the average soil concentration, the biodegradation flag is an important input parameter, and the biodegradation rate constants used in the model are also critical parameters. The first-order biodegradation rate was included for all LAU runs and for the long-term wastepile emission runs; biodegradation losses were not included in the short-term wastepile emission runs or any of the landfill runs, because these units are not designed specifically for biodegradation and because of the relatively short-time period for which the waste was assumed to be in the unit. Biodegradation was included for wastepiles as the median retention time of waste in the wastepiles is greater than 5 years. Note that the default CHEMDAT8 model method of calculating the biodegradation rate was not used because the biodegradation rates used in CHEMDAT8 were derived primarily from wastewater studies and then applied with an assumed, low-biomass concentration to model biodegradation in soils.

Instead, the first-order biodegradation rate constants developed for the HWIR project based, for the most part, on contaminant half lives in soils were used. Therefore, the HWIR biodegradation rate constants provide a more direct link to soil-based biodegradation and are considered more appropriate for all land-based WMUs that include biodegradation.

4.2.2.2 Particulate Emissions. Particulate emissions due to wind erosion were modeled for land-based units (landfills, land application units, and wastepiles). Landfills and LAUs were modeled differently than wastepiles because they are ground-level sources and wastepiles are elevated sources. Wind erosion emissions from landfills and LAUs were modeled using the Cowherd model (U.S. EPA, 1985b and U.S. EPA, 1988). This model estimates the emission of respirable particles (i.e., PM_{10}) due to wind erosion from a ground-level surface with an unlimited reservoir of erodible particles. Surfaces are defined as having a limited or unlimited reservoir based on threshold friction velocity (U^*); surfaces with a U^* greater than 0.5 m/s are considered limited; those with U^* less than 0.5 m/s are considered unlimited (U.S. EPA, 1988). Threshold friction velocity is a measure of the windspeed at the ground surface that would be required to remove particles from the surface. Examples of limited reservoirs include nonhomogeneous surfaces with stones, clumps of vegetation, or other nonerodible elements or crusted surfaces. Further, wind erosion is considered unlikely to occur from surfaces with full vegetative cover.

Wind erosion emissions from wastepiles were modeled using an equation from AP-42 (U.S. EPA, 1985a) for estimating emissions from wind erosion from active storage piles. The equation gives emissions of total suspended particulates (TSP). Typically, an equation-specific particle size multiplier would be applied to reduce the emissions to a desired size category, in this case, PM_{10} . No particle size multipliers are given for this equation in AP-42; however, Cowherd (U.S. EPA, 1988) gives a PM_{10} particle size multiplier of 0.5 for use with this equation.

Important input parameters for this calculation include silt content of waste (i.e., percent with small particle size), number of days with greater than 0.01 inches of rainfall, and percent of time that windspeed exceeds 5.4 m/s. Data on the silt content of the wastes being modeled were not available. A median silt content for miscellaneous fill material of 12 percent (U.S. EPA, 1988) was used. The number of precipitation days and the frequency of windspeed greater than 5.4 m/s were location-specific; values were obtained from NOAA (1992) and are summarized in Section 4.7.2.

4.2.3 Critical Parameters for Tank Emissions Model

The input parameters for aerated tanks are presented in Table 4-2. The annual waste quantity (flow rate) and the dimensions of the tank are critical input parameters for both aerated and storage tanks. Unlike the other WMUs, site-specific / unit-specific data were not available for tanks. Therefore, the flow rate and dimensions of the tanks must be estimated. Two model tanks that have been used previously by EPA to assess emission from aerated tanks were used for this analysis for both aerated and storage tanks (U.S. EPA, 1991).

Factors that impact the relative surface area of turbulence and the intensity of that turbulence are important factors in determining the fate of chemicals in aerated tanks. The

Table 4-2. CHEMDAT8 Tank Model Input Requirements

Input Parameter	Data Source/Assumption
For both aerated and storage tanks:	
Windspeed (m/s)	Set by location of WMU
Temperature (°C)	Set by location of WMU
Depth (m)	Input based on model unit ^a
Area (m ²)	Input based on model unit ^a
Flow rate (m ³ /s)	Input based on model unit ^a
Active biomass (g/L)	With biodegradation: set to 2 for aerated tanks, set to 0.05 for storage tanks Without biodegradation: set to 0.
Biomass solids in (g/L)	Assumed value of 0
VO inlet conc. (mg/L)	Unit conc. assumed - set to 1
Total organics in (mg/L)	Assumed value of 100
Total biorate (mg/g bio-h)	Assumed value of 19
For aerated tanks only:	
Fraction agitated	Input based on model unit ^a
Submerged air flow (m ³ /s)	Set to 0 (mechanical aeration)
Number impellers	Input based on model unit ^a
Oxygen trans. rating (lbO ₂ /h-HP)	Input based on model unit ^a = 3
Power (total)(HP)	Input based on model unit ^a
Power efficiency	Input based on model unit ^a = 0.83
Impeller diameter (cm)	Input based on model unit ^a = 61
Impeller speed (cm)	Input based on model unit ^a = 130

^a Two model units were run at each meteorological location both with and without active biodegradation. The input parameters for the two model tanks were developed by EPA in the development of emission standards for hazardous wastes (U.S. EPA, 1991). Set to 0 for storage tanks.

aerated tank model has several input parameters that impact the degree and intensity of the turbulence created by the aeration (or mixing). These input parameters include Input ID Nos. T10, T11, T12, T13, T14, T15, T17 and T18. The aerated tank model is most sensitive to the fraction aerated; the total power, power per aerator (number of aerators), and impeller diameter have some impact on the emission results; and the other parameters have little to no impact on the estimated emissions (see Appendix C). The values of most of these parameters were selected from the model aerated tank parameters developed by EPA (U.S. EPA, 1991). Submerged air flow was set to zero (assumed mechanical aeration only).

Factors that influence the rate of biodegradation are important in determining emissions from both aerated and storage tanks. Unlike the biodegradation rate model that was used for the land-based units, the biodegradation rate model used in CHEMDAT8 for tanks is dependent on the amount of active biomass in the WMU. Therefore, the active biomass concentration (Input ID No. T5) is a critical parameter for aerated and storage tanks. Because this parameter can vary

widely for different types of tanks, duplicate model runs were performed at two different biomass concentrations for both aerated and storage tanks. For both aerated and storage tanks, one model run was made using a biomass concentration of zero (no biodegradation) and one model run was made that included biodegradation. A zero biomass concentration is appropriate for short-term storage tanks or for aerated tanks designed primarily for mixing, but not for enhanced biodegradation units. Examples of tanks that might be actively mixed (aerated) but have little or no biodegradation are equalization basins and neutralization tanks. For the second aerated tank run, the CHEMDAT8 aerated tank default value of 2.0 g/L was used for the biomass concentration. This biomass concentration is appropriate for aerated tanks designed for enhanced biodegradation (e.g., activated sludge wastewater treatment tanks). For the second storage tank run, a biomass concentration of 0.05 g/L was selected. This value is appropriate for wastewater treatment tanks that are not specifically designed for biodegradation but where it may occur passively.

Due to the nonlinearity of the biodegradation rate model used in the tank emission estimates, direct backcalculation of an acceptable waste concentration may not be appropriate for some compounds. Unlike the emission results from the land-based units, the contaminant ("VO inlet") concentration used in the analysis may impact the predicted "normalized" emission rate (i.e., the emission rate in $\text{g/m}^2\text{-s}$ per mg/L of contaminant). Therefore, the model tanks were run at a low unit concentration (i.e., 1 mg/L) and at a high concentration (i.e., the constituent's solubility). The most appropriate backcalculated emission value was then selected based on the concentration range of the backcalculated values and the constituent's biodegradation characteristics (see Section 7.9 for further details).

Meteorological inputs are also important for the tank emission model. For the storage tank, the emission estimates are impacted by both temperature and windspeed. As the emissions for the aerated tank are predominantly driven by the turbulent area and associated mass transfer coefficients, the emissions from the aerated tank are not strongly impacted by the windspeed. Aerated tank emissions are impacted by temperature. Note that, dependent on the residence time of the waste in the tank, the temperature of the waste in the tank is not expected to vary significantly with changing atmospheric temperatures, and annual average temperatures were used to estimate the average waste temperature in the tanks. Each model tank was run for each of the 29 meteorological regions used in the analysis for each of the model assumptions (aerated and nonaerated, with biodegradation and without biodegradation, and high concentration and low concentration).

The "biomass solids in," "total organics in," and "total biorate" (Input ID Nos. T6, T8, and T9) impact the rate of biomass production and subsequently the amount of contaminant that is absorbed onto the solids. (Note: The "biomass solids in" does not affect the biodegradation rate and is more appropriately labeled simply "solids in. "). These inputs, however, have little or no impact on the estimated emission rates for most of the contaminants modeled in this analysis. The CHEMDAT8 default value for solids in is zero, and this value was used for all aerated tank model runs. The CHEMDAT8 default values for contaminant concentration ("VO inlet conc.") and total organics concentration are 100 and 250 mg/L , respectively. Because a unit contaminant concentration of 1 mg/L was used, it seemed appropriate to similarly reduce the total organics

concentration. Therefore, the total organics input was set at 100 mg/L. The CHEMDAT8 default value of 19 mg/g-h for "total biorate" was used for the tank model runs.

4.3 Development of Volatile Emissions and Waste Concentrations for Landfills

The basic assumptions used for modeling landfills are as follows:

- The landfill operates for 20 years filling 20 equal cells sequentially.
- The active cell is modeled as being instantaneously filled at time $t=0$ and remains open for 1 year.
- Emissions are calculated only for one cell for 1 year (either the cells are depleted of the constituent or capped after 1 year).
- The waste is homogeneous with an initial concentration of 1 mg/kg.
- The waste matrix is aqueous (Henry's law partitioning applies).
- Annual average temperature is used (determined by assigned meteorological station).
- Acute and subchronic exposures were not modeled.

Emissions based on the aqueous waste matrix assumption sometimes yielded backcalculated concentration values that exceeded the soil saturation level for certain compounds. Therefore, an additional model run was made for landfills using a concentration of waste of $1\text{E}+6$ mg/kg (pure component) and turning off the aqueous waste flag (i.e., employed Raoult's law partitioning). These model runs provided a means to check the appropriateness of backcalculated concentration, and they also provide a more environmentally conservative backcalculated concentration estimate for constituents with high vapor pressure but low Henry's law constants (e.g., formaldehyde).

Table 4-1 provides the CHEMDAT8 model input requirements for land-based units with some commentary about each input parameter. The inputs that were calculated from the Industrial D Screening Survey data were calculated as follows:

1. All total quantities, capacities, and areas in the Industrial D Screening Survey were divided by the number of landfills at the facility to get landfill-specific estimates.
2. Loading = bulk density = 1.09577 g/cm^3 .

3. Tilling depth (cm) = landfill depth, l , calculated as follows:

$$l \text{ (cm)} = \frac{100 \text{ (cm/m)} \times [\text{Capacity (Mg)} \times 1\text{E}+6 \text{ (g/Mg)}]}{[\text{Area (acre)} \times 4047 \text{ (m}^2\text{/acre)} \times \text{Bulk Density (g/cm}^3\text{)} \times (100 \text{ cm/m)}^3]} \quad (4-1)$$

If the calculated depth was less than 50 cm or more than 832 cm, then the method described in Section 3.1 was used.

4. Total landfill surface area was divided by 20 to get surface area of landfill cell.
5. The total landfill capacity was divided by 20 to get the average annual quantity of waste, Q_{annual} .

The landfill *cell* areas and depth were entered into the CHEMDAT8 input table (along with average ambient temperature), and the emission fraction (in Column AU - "total emissions" output for the "intermediate time" [365.25 days]) was calculated. This emission fraction was then multiplied by the annual waste quantity and waste concentration and divided by the area of the cell to calculate the output emission rate as follows:

$$\text{Emiss.Rate (g/m}^2\text{-s)} = \frac{Q_{\text{annual}}(\text{Mg/yr}) \times C_{\text{waste}}(\text{g/Mg}) \times \text{Emiss.Fract.}}{\text{Area}_{\text{cell}}(\text{m}^2) \times 365.25 \times 24 \times 3600(\text{s/yr})} \quad (4-2)$$

The average concentration of the waste in the landfill cell was estimated from the emission fraction and the biodegradation fraction (although the biodegradation fraction was zero – no biodegradation – for the landfill) by assuming first-order contaminant (concentration) disappearance. Assuming first-order kinetics with respect to the contaminant concentration in the landfill cell, an exponential decay can be written in terms of the apparent overall first-order decay rate. The concentration at a given time is equal to the initial concentration as follows:

$$C_{\text{waste},t} = C_{\text{waste},o} \times \exp(-K_{1,\text{all}} t) \quad (4-3)$$

At the end of 1 year, $C_{\text{waste},t}/C_{\text{waste},o} = 1 - \text{emiss.fract.} - \text{biodegr.fract.}$. Therefore, the $K_{1,\text{all}} t$ term, at the time period for which the fraction loss terms were calculated, is simply:

$$K_{1,\text{all}} t = -\ln(1 - \text{emiss.fract.} - \text{biodegr.fract.}) \quad (4-4)$$

The concentration versus time profile (Equation 4-3) can then be integrated to calculate the average waste concentration, $C_{\text{waste,ave}}$, over the time period of the calculation:

$$C_{waste,ave} = C_{waste,o} \left(\frac{[1 - \exp(-K_{1,all} t)]}{K_{1,all} t} \right). \quad (4-5)$$

Because columns AW and AX in the CHEMDAT8 spreadsheet model were not needed for the landfill runs, Equations 4-4 and 4-5 were entered in these columns to calculate the average output soil concentrations present in the active landfill cell.

The input parameters required for the landfill are presented in Table 4-1. The aqueous waste flag is set to 1 (the most critical model flag), and the annual waste quantity and unit dimensions are the critical source parameters. For landfills, the loading rate is pure waste material so that Input ID No. L1 is basically the waste density. A waste density of 1.09577 g/cm³ was used for the landfill to be consistent with the waste densities used in the analysis of the Industrial D Screening survey data. The annual waste quantity is also combined with the area of the landfill to calculate the depth of the landfill.

Temperature and porosities have some impact on predicted emissions. The biodegradation flag was set to zero (no biodegradation) for landfills. Therefore, temperature variations should have less of an impact on the annual emission rates from landfills than from land application units. The model is insensitive to molecular weight of the waste (for aqueous wastes) and windspeed (for long-term emission estimates).

4.4 Development of Volatile Emissions and Waste Concentrations for Land Application Units

4.4.1 Chronic Exposure Analysis

Because the same basic CHEMDAT8 model was used for landfills and land application units, the emissions estimates for land application units have some similarities to the landfill emission estimates, but there are a number of differences. The basic modeling assumptions used for modeling land application units are as follows:

- The land application unit operates at pseudo-steady-state (pseudo-steady-state may be reached in 1 to 2 years or as time approaches infinity, depending on the application and loss rates).
- Waste application occurs quarterly.
- The waste is homogeneous with an initial concentration of 1 mg/kg.
- The waste matrix is aqueous (Henry's law partitioning applies).
- Temperature is determined by assigned meteorological station; quarterly average temperature was used.

- Biodegradation occurs at temperatures greater than 5 °C.

Emissions based on the aqueous waste matrix assumption sometimes yielded backcalculated concentration values that exceeded the soil saturation level for some compounds. Therefore, an additional model run was made for LAU using a concentration of waste of 1E+6 mg/kg (pure component) and turning off the aqueous waste flag (i.e., employed Raoult's law partitioning). These model runs provided a means to check the appropriateness of backcalculated concentration, and they also provide a more environmentally conservative backcalculated concentration estimate for constituents with high vapor pressure but low Henry's law constants (e.g., formaldehyde).

The inputs that were calculated for the land application units were calculated as follows:

1. The total annual waste quantities and surface areas for each facility, as reported in the Industrial D Screening Survey, were divided by the number of land application units at the facility to get land application unit-specific estimates.
2. Tilling depth (cm) = 20 cm if $Q_{\text{annual}} \text{ (Mg/yr) / Area (m}^2\text{)} \leq 0.2$. If $Q_{\text{annual}} / \text{Area} > 0.2$, then depth (cm) = $100 \times Q_{\text{annual}} / \text{Area}$.
3. Loading rate, L , is calculated as follows:

$$L \text{ (g/cm}^3\text{)} = \frac{Q_{\text{annual}} \times 0.25 \text{ yr}}{\text{Area} \times (\text{depth}/100)} \quad (4-6)$$

4. Biodegradation flag input (Input ID No. L9) = 1 if temperature is greater than 5 °C. If the temperature is 5 °C or lower, the biodegradation flag input = 0.
5. Time of calc. = $365.25/4 = 91.31$ days.
6. Quarterly/seasonal temperature and windspeeds were calculated by averaging the monthly temperature and windspeeds using the following groupings by month:
 - Winter: December through February
 - Spring: March through May
 - Summer: June through August
 - Fall: September through November.

The CHEMDAT8 model was run for each land application for each of the four seasons. Equation 4-4 was used to estimate $K_{i,\text{all}} t$ for a given season. However, for the land application unit, additional waste input occurs after the modeled time step. It is assumed that the volume of the land application unit remains constant. Therefore, as more waste is applied, it is assumed that an equal volume of waste/soil mixture becomes buried or otherwise removed from the active tilling depth. At pseudo-steady-state (approaching infinite time), the total contaminant mass lost

from the system equals the total contaminant mass added to the system. Including the contaminant loss by burial/removal, the initial soil concentration at the pseudo-steady-state is

$$C_{o,ss} \text{ (g/Mg)} = \frac{C_{waste,o} \times \left(\frac{L}{Bulk \text{ Dens.}} \right)}{1 - \left[\exp(-K_{1,all} t) \times \left(1 - \frac{L}{Bulk \text{ Dens.}} \right) \right]} \quad (4-7)$$

The average pseudo-steady-state concentration over the time interval of the calculation, in this case 91.31 days, is calculated using Equation 4-5, but using $C_{o,ss}$ as calculated in Equation 4-7 in the place of $C_{waste,o}$. In this manner, the long-term average concentration for a given season was calculated. Once the long-term average concentration was calculated for all four seasons, the arithmetic average of these seasonal concentrations was calculated and output.

The emission fraction (in Column AU - "total emissions" output for the "intermediate time" [91.31 days]) was multiplied by the pseudo-steady-state initial soil concentration (from Equation 4-7), the bulk density, and the tilling depth and divided by the time period of the calculation to determine the long-term average emission rate for that season as follows:

$$Emiss.Rate \text{ (g/m}^2\text{-s)} = \frac{C_{o,ss} \times Emiss.Fract. \times Bulk \text{ Dens.} \times (L_{till}/100)}{Time \text{ Calc.}(d) \times [24 \times 3600(s/d)]} \quad (4-8)$$

The long-term average seasonal emission rates were calculated for all four seasons, then the arithmetic average of these seasonal emission rates was calculated and output. The input parameters required for the land application unit are presented in Table 4-1. The aqueous waste flag is set to 1 (the most critical model flag). The annual waste quantity and unit dimensions are the critical source parameter. The area of the land application unit is a site-specific input parameter. The tilling depth was assumed to be 20 cm unless the annual quantity of waste added to the land application unit was greater than the volume of the land application unit with a set depth of 20 cm. If the annual waste quantity exceeded the volume of the land application unit given a depth of 20 cm, the depth of the land application unit was calculated so that the land application unit can hold the entire annual waste quantity. Quarterly waste applications were employed (Input ID No. L8 was set to 91.3 days) so that any given quarterly waste application occupied no more than 25 percent of the land application unit.

A waste density of 1.09577 g/cm³ was used to test the unit size and to calculate the tilling depth, if needed. This waste density was used for the land application unit to be consistent with the waste densities used in the analysis of the Industrial D Screening survey data. The air porosity has the greatest influence on the predicted emissions of these parameters. Nonetheless, as these parameters typically do not vary over wide ranges, these parameters are considered secondary parameters for the emissions estimates.

Because biodegradation both lowers the emissions rate and the average soil concentration, the biodegradation flag is an important input parameter, and the biodegradation rate constants used in the model are also critical parameters. The biodegradation flag was set to 1 for all land application unit runs; however, the default CHEMDAT8 model method of calculating the biodegradation rate was not used. Instead, the HWIR values (RTI, 1995) for first-order biodegradation rate constants were used. The HWIR biodegradation rate constants are based, for the most part, on contaminant half-lives in soil. The biodegradation rates used in CHEMDAT8 were derived primarily from wastewater studies, and then a low biomass concentration was used in CHEMDAT8 to model biodegradation in soils. Because the HWIR biodegradation rate constants were more directly linked to soil-based WMUs, the HWIR biodegradation rate constants were used for all land-based WMUs that included biodegradation.

4.4.2 Acute and Subchronic Exposure Analysis

Short-term (1-day and 1-month) emission estimates were made for LAUs. These emission estimates used the same general modeling approach as used for the chronic approach; however, the emission estimates were made for the first 24 hours or first 30 days immediately after a waste application (subsequent to reaching steady state). As with the chronic exposure emission estimates, biodegradation was included in the model.

The short-term emission estimates were projected from the CHEMDAT8 "instantaneous" emission values by weighting the instantaneous emission rate with a corresponding time interval (see box).

In the absence of biodegradation, higher temperatures would produce higher volatile emissions. However, when biodegradation is modeled, it slows to zero at temperatures below 5°C, thus increasing volatile emissions at low temperatures. In attempts to develop reasonable worst-case emission estimates, each LAU was run at a high and low temperature. The high temperature used was the highest average monthly temperature for the assigned meteorological location. The low temperature used was either the lowest average monthly temperature or 5°C, whichever was higher. Because the acute (1-day) emission estimates are dependent on windspeed, and higher windspeeds provide higher emissions, the windspeed used for the acute emission estimate was the annual average windspeed for the assigned meteorological location multiplied by 1.5 to estimate a reasonable worst-case windspeed. The higher of the emission rates calculated from the high and low temperature runs was used as input into the exposure model. Average soil concentrations corresponding to the emission estimates were also output for the exposure model.

Time at Which Instantaneous Emissions were Calculated	Period of Time Represented by Instantaneous Emissions
0.25 h	0 - 0.5 h
1 h	0.5 - 2 h
4 h	2 - 6 h
10 h	6 - 14 h
18 h	14 - 24 h

Acute and subchronic emission estimates were made for both unit and pure component concentrations. The Taylor series approximation used to estimate short-term (i.e., subchronic) emissions in CHEMDAT8 is inaccurate for chemicals of very low volatility. For six chemicals with the lowest Henry's law constants, this resulted in subchronic emissions greater than acute emissions (which are instantaneous rates based on an exact solution rather than the Taylor series approximation). For chemicals with very low volatility, the instantaneous emission rates are not expected to change much over time. Therefore, for these six chemicals, the acute (instantaneous) emission rates and soil concentrations were also used for subchronic exposure estimates.

4.5 Development of Volatile Emissions and Waste Concentrations for Wastepiles

4.5.1 Chronic Exposure Analysis

Again, the same basic CHEMDAT8 model that was used for landfills and land application units was also used for wastepiles. Similar to landfills, waste is applied, and the basic modeling equations used for landfills apply. However, subsequent calculations of the wastepile results are required to account for "seasonal" variations, similar to the land application units. The basic modeling assumptions used for modeling land application units are as follows:

- The wastepile operates with a fixed volume; fixed depths of 2 and 5 meters were assumed; all wastepiles were modeled at both assumed depths.
- The waste is homogeneous with an initial concentration of 1 mg/kg.
- The waste matrix is aqueous (Henry's law partitioning applies).
- Quarterly average temperatures are used; temperature determined by assigned meteorological station.
- Maximum monthly temperature was used for acute and subchronic exposures.
- Biodegradation occurs at temperatures greater than 5 °C.
- No biodegradation was assumed for acute and subchronic exposures.

Emissions based on the aqueous waste matrix assumption sometimes yielded backcalculated concentration values that exceeded the soil saturation level for some compounds. Therefore, an additional model run was made for wastepiles using a concentration of waste of 1E+6 mg/kg (pure component) and turning off the aqueous waste flag (i.e., employed Raoult's law partitioning). These model runs provided a means to check the appropriateness of back-calculated concentration, and they also provide a more environmentally conservative back-calculated concentration estimate for constituents with high vapor pressure but low Henry's law constants (e.g., formaldehyde).

Inputs that were calculated for the wastepile were calculated as follows:

1. The total annual waste quantities and surface areas for each facility, as reported in the Industrial D Screening Survey, were divided by the number of wastepiles at the facility to get wastepile unit-specific estimates.
2. Loading = waste bulk density = 1.09577 g/cm³.
3. Tilling depth (cm) = 200 and 500 (all units run at both depths).
4. Biodegradation flag input (Input ID No. L9) = 1 if temperature is greater than 5 °C. If the temperature is 5 °C or lower, the biodegradation flag input = 0.
5. Time of calc. = average residence time of waste in the wastepile, t_{ave} , as follows:

$$Time\ Calc.(d) = \frac{Area \times (l_{till}/100) \times Bulk\ Dens. \times 365.25(d/yr)}{Q_{annual} (Mg/yr)} \quad (4-9)$$

6. The same quarterly/seasonal temperature and windspeeds calculated for land application units were used for wastepiles.

The average waste concentration and emission rate for the wastepile can be calculated using the equations presented for the landfill model (Equations 4-2 and 4-5). (Note: Because the loading rate equals the bulk density, Equation 4-7 reduces to $C_{o,ss} = C_{o,waste}$, and the equations used for land application units could also be used; substitution of Equation 4-9 into 4-8 yields Equation 4-2). Due to the extremely long residence times calculated for some of the wastepiles, an if-statement was required when calculating $(K_{1,all} t)$ to prevent taking the logarithm of zero. The model was found to truncate when: $1 - emiss.fract. - biodegr.fract < 10E-15$.

The same input parameters required for land application units and landfills were used for wastepiles (see Table 4-1). The aqueous waste flag is set to 1 (the most critical model flag). The annual waste quantity and unit dimensions are still the critical source parameter. As with landfills, the loading for wastepiles is pure waste material so that Input ID No. L1 is basically the waste density. A waste density of 1.09577 g/cm³ was used for the wastepile to be consistent with the waste densities used in the analysis of the Industrial D Screening survey data. Wastepile emission (and concentration) estimates were made at two set wastepile depths. The annual waste quantity combined with the capacity (dimensions) of the wastepile were used to calculate residence time for the waste in the wastepile (i.e., time of calculation - Input ID No. L8).

Temperature and porosities continue to have some impact on emissions. The biodegradation flag was set to 1 (biodegradation included) for wastepiles. The model remains insensitive to molecular weight of the waste (for aqueous wastes) and windspeed (for long-term emission estimates).

4.5.2 Acute and Subchronic Exposure Analysis

Acute (1-day) and subchronic (1-month) emission estimates were made for wastepiles following the same approach used for LAU as described in Section 4.4.2, with the following differences:

- The wastepile was assumed to be completely filled with new waste at time $t = 0$.
- Biodegradation was **not** included (a lag period is generally associated with biodegradation, especially for the assumed "all new waste" scenario).
- Since there was no biodegradation, only high-temperature runs were needed.

4.6 Development of Volatile Emissions for Tanks

The only output for tanks is the annual air emissions. Consequently, additional equations did not have to be added to the CHEMDAT8 model to run the tank model as was needed for the land-based WMUs. However, for the aerated tank emission estimates, a number of if-statements were needed to prevent division by zero for chemicals that did not have biodegradation rate constants, vapor pressure, Henry's law constant, or diffusivity inputs (e.g., metals). These if-statements set the ultimate biodegradation rate or volatilization rate to zero when the biodegradation rate or volatilization rate inputs were either zero or missing.

The basic modeling assumptions used (or inherent in CHEMDAT8) for the aerated tank model emission estimates include:

- The WMU operates at steady state.
- The WMU is well mixed.
- Emission estimates were performed for two different influent concentrations: an influent concentration of 1 mg/L (i.e., "VO Inlet conc." input = $C_{\text{infl}} = 1 \text{ mg/L} = 1 \text{ g/m}^3$) and an influent concentration equal to the constituent's solubility.
- The waste matrix is aqueous (Henry's law partitioning applies).
- Annual average temperatures are used; temperature determined by assigned meteorological stations.
- Biodegradation rate is first order with respect to biomass concentrations.
- Biodegradation rate follows Monod kinetics with respect to contaminant concentrations.
- Hydrolysis rate is first order with respect to contaminant concentrations.

- Acute and subchronic exposures were not modeled.

Emissions based on the aqueous waste matrix assumption sometimes yielded backcalculated concentration values that exceeded the aqueous solubility level for some compounds. Therefore, additional model runs were made using a concentration of waste of 1E+6 mg/L (pure component) and modifying the CHEMDAT8 model equation for the dimensionless Henry's law constant to be the constituent's partial vapor pressure (i.e., to employ Raoult's law partitioning). These model runs provided a means to check the appropriateness of backcalculated concentration values higher than the solubility limit. It also provided a more environmentally conservative backcalculated concentration estimate for constituents with high vapor pressure but low Henry's law constants (e.g., formaldehyde).

For tanks, the surface area, depth, and flow rate are all directly specified by the model units. Two model units were run: a small tank and a large tank. The emissions from these units were modeled at all 29 of the selected meteorological regions, both with and without biomass (i.e., 2 units \times 2 biomass conditions \times 29 meteorological locations \times 2 concentrations = 232 runs). The storage tank emission estimates were performed using the same model tanks that were used for aerated tanks, but the fraction agitated (T10) was set to zero.

The CHEMDAT8 model is used to calculate the emission fractions for the model units. The emission rate, in g/m²-s, is calculated from the fraction emitted, the flow rate, waste concentration, and the surface area as follows:

$$Emiss.Rate \text{ (g/m}^2\text{-s)} = \frac{Q_{flow} \text{ (m}^3\text{/s)} \times C_{infl} \text{ (g/m}^3\text{)} \times Emiss.Fract.}{Area \text{ (m}^2\text{)}} \quad (4-10)$$

4.7 Development of Particulate Emissions

Two different models were used to model wind erosion – one for wastepiles (elevated sources) and one for landfills and land application units (ground-level sources). The Cowherd model (U.S. EPA, 1985b and 1988) was selected for modeling wind erosion emissions from ground-level sources, and the AP-42 model for wind erosion from aggregate storage piles (U.S. EPA, 1985a) was selected for modeling wind erosion emissions from wastepiles.

For both types of WMU, the models described in this section predict the emission rate of particulate matter released from a site due to wind erosion. To obtain the emission rate of constituent sorbed to particulate matter, the emission rate of particulate matter must be multiplied by the soil or waste concentration. That calculation is described in Section 7.4.

4.7.1 Landfills and Land Application Units

Wind erosion emissions from landfills and LAUs were modeled using the Cowherd model (U.S. EPA, 1985b). A newer version of Cowherd's model is available (U.S. EPA, 1988). However, this is an event-based model that requires detailed site-specific information unavailable

for this analysis. Therefore, it was not used. The older Cowherd model tends to overestimate emissions relative to the event-based version. Although the degree to which it overestimates is not known, it is expected to be relatively small. Since particulate emissions are negligible compared to volatile emissions for the volatile chemicals modeled, this is only of concern for the metals (other than mercury), which are based only on particulate emissions.

The Cowherd model estimates the emission of respirable particles (i.e., PM_{10}) due to wind erosion from a ground-level surface with an unlimited reservoir of erodible particles. Surfaces are defined as having a limited or unlimited reservoir based on threshold friction velocity (U^*); surfaces with a U^* greater than 0.5 m/s are considered limited, while those with U^* less than 0.5 m/s are considered unlimited (U.S. EPA, 1988). Threshold friction velocity is a measure of the windspeed at the ground surface that would be required to remove particles from the surface. Examples of limited reservoirs would include nonhomogeneous surfaces with stones, clumps of vegetation, or other nonerodible elements or crusted surfaces. Further, wind erosion is considered unlikely to occur from surfaces with full vegetative cover.

Wind erosion emissions were calculated as follows (U.S. EPA, 1985b):

$$E_{10} = 0.036 \times (1-V) \times \left(\frac{[u]}{U_t} \right)^3 \times F(x) \quad (4-11)$$

where

- E_{10} = emission rate of PM_{10} (g/m^2-h)
- V = vegetative cover (fraction)
- $[u]$ = average annual windspeed (m/s)
- U_t = threshold windspeed (m/s).

$F(x)$ is a complex function of x , which is a ratio of threshold windspeed and average annual windspeed. The following is a linear approximation of $F(x)$ from a graph of $F(x)$ in U.S. EPA (1985b, Figure 4-3), except for the equation for values of x greater than 2, which is given in Appendix B of U.S. EPA (1985b).

$$F(x) = \begin{array}{ll} 1.91 & x \leq 0.5 \\ 2.06 - 0.33x & 0.5 < x < 0.8 \\ 2.6 - x & 0.8 < x < 1 \\ 2.9 - 1.3x & 1 < x < 2 \\ 0.18(8x^3 + 12x)e^{-x^2} & x \geq 2 \end{array} \quad (4-12)$$

$$x = 0.886 \times \frac{U_t}{[u]} \quad (4-13)$$

The threshold windspeed, U_t , is a function of threshold friction velocity and roughness height, as follows:

$$U_t = \frac{1}{0.4} \times \ln\left(\frac{Z}{z_0}\right) \times U^* \quad (4-14)$$

where

- U_t = threshold windspeed (m/s)
- Z = anemometer height (cm)
- z_0 = roughness height (cm)
- U^* = threshold friction velocity (m/s).

The inputs used for these equations (other than average annual windspeed and anemometer height, which are location-specific) are summarized in Table 4-3.

The vegetative cover, V , is an important modifying factor that can range from 0 (bare ground) to 1 (100 percent vegetative cover). Because the surface being modeled is either an active landfill cell or an active LAU, it seemed reasonable to assume that no vegetation would have the opportunity to grow on the surface of the WMU while it was active. Therefore, vegetative cover was set to 0.

The roughness height, z_0 , accounts for the size and spacing of surface roughness elements (which may include vegetation, or even buildings). U.S. EPA (1985b) provides a range of roughness heights for various surfaces ranging from 0.1 cm (natural snow) to 1,000 cm (high-rise buildings). A value of 1 cm, corresponding to a plowed field, was selected as most representative of the conditions expected to occur in an active landfill or LAU.

The threshold friction velocity, U^* , is a function of the physical condition of the soil and would normally be considered a site-specific parameter. In the absence of data, a value of 0.5 m/s was selected based on the assumption that the WMU is an unlimited reservoir.

Table 4-3. Inputs and Intermediate Values Used for Wind Erosion from Landfills and LAUs

Symbol	Parameter	Units	Value	Source
V	Vegetative cover	fraction	0	Assumption
z_0	Roughness height	cm	1	U.S. EPA (1985b)
U^*	Threshold friction velocity	m/s	0.5	Assumption

The following inputs and intermediate values are location-specific: anemometer height, threshold windspeed, average annual windspeed, x , and $F(x)$. These are shown in Table 4-4 along with the calculated particulate emissions rate for each meteorological location used in the analysis. The anemometer heights and average annual windspeed values are from NOAA (1992). Values for u_t , x , $F(x)$, and E_{10} have been rounded to two significant figures.

4.7.2 Wastepiles

Wind erosion emissions from wastepiles were modeled using an equation from AP-42 (U.S. EPA, 1985a) for estimating emissions from wind erosion from active storage piles. The equation gives emissions of total suspended particulates. Typically, an equation-specific particle size multiplier would be applied to reduce the emissions to a desired size category, in this case, PM_{10} . No particle size multipliers are given for this equation in AP-42; however, Cowherd (U.S. EPA, 1988) gives a PM_{10} particle size multiplier of 0.5 for use with this equation.

Table 4-4. Calculated Particulate Emission Rates for Landfills and LAUs

Location	Z (cm)	U_t (m/s)	u (m/s)	x (unitless)	$F(x)$ (unitless)	E_{10} (g/m ² -h)
Albuquerque	700	8.2	4.1	1.8	0.06	0.0027
Atlanta	610	8.0	4.6	1.5	0.89	0.0061
Bismarck	610	8.0	6.2	1.1	1.4	0.023
Boise	610	8.0	4.6	1.5	0.89	0.0061
Casper	610	8.6	7.2	1.1	1.5	0.032
Charleston	610	8.0	4.1	1.7	0.65	0.0031
Chicago	610	8.0	4.6	1.5	0.89	0.0061
Cleveland	1010	8.6	5.1	1.5	0.95	0.0070
Denver	1010	8.6	4.1	1.9	0.47	0.0018
Fresno	610	8.0	3.6	2.0	0.34	0.0011
Harrisburg	610	8.1	4.6	1.6	0.86	0.0056
Hartford	1010	8.6	4.1	1.9	0.47	0.0018
Houston	610	8.0	4.1	1.7	0.65	0.0031
Huntington	610	8.0	3.6	2.0	0.34	0.0011
Las Vegas	610	8.0	5.1	1.4	1.1	0.010
Lincoln	610	8.0	5.1	1.4	1.1	0.010
Little Rock	610	8.0	3.6	2.0	0.34	0.0011
Los Angeles	910	8.5	4.1	1.8	0.51	0.0020
Miami	700	8.2	4.6	1.6	0.85	0.0054
Minneapolis	1010	8.6	5.7	1.3	1.2	0.012
Philadelphia	610	8.0	4.6	1.5	0.89	0.0061
Phoenix	1010	8.6	3.1	2.5	0.060	0.00010
Portland, ME	610	8.0	4.6	1.5	0.89	0.0061
Raleigh-Durham	610	8.0	4.1	1.7	0.65	0.0031
Salem, OR	610	8.0	4.6	1.5	0.89	0.0061
Salt Lake City	610	8.0	4.6	1.5	0.89	0.0061
San Francisco	1010	8.6	6.2	1.2	1.3	0.017
Seattle	610	8.0	5.1	1.4	1.1	0.010
Winnemucca	1010	8.6	4.1	1.9	0.47	0.0018

Emissions were calculated as follows:

$$E_{10} = 1.9 \times PSM \times \frac{S}{1.5} \times \frac{365-p}{235} \times \frac{f}{15} \quad (4-15)$$

where

- E_{10} = emission rate of PM_{10} (kg/ha-d)
- S = silt content of waste (%)
- p = number of days with ≥ 0.25 mm (0.01 in) of precipitation per year (d/yr)
- f = percentage of time that the unobstructed windspeed exceeds 5.4 m/s (12 mph) (%)
- PSM = particle size multiplier for PM_{10} (unitless) = 0.5.

The emission rate was converted to $g/m^2\cdot h$ as follows:

$$E(g/m^2\cdot h) = E(kg/ha\cdot d) \times \frac{1,000 g/kg}{10,000 m^2/ha \times 24 h/d} \quad (4-16)$$

Data on the silt content of the wastes being modeled were not available. As a conservative assumption, the silt content of miscellaneous fill materials from AP-42 was used. This value is 12 percent.

The number of precipitation days and the frequency of windspeed greater than 5.4 m/s were location specific; values were obtained from NOAA (1992) and are summarized in Table 4-5. Also included in Table 4-5 are the calculated particulate emission rates in both kg/ha-d and $g/m^2\cdot h$; these values have been rounded to two significant figures.

Table 4-5. Calculated Particulate Emission Rates for Wastepiles

Location	p (d/yr)	f (%)	E (kg/ha-d)	E (g/m ² -h)
Albuquerque	58	22.0	15	0.061
Atlanta	116	21.0	11	0.047
Bismarck	96	33.2	19	0.080
Boise	91	21.0	12	0.052
Casper	95	47.3	28	0.11
Charleston	113	19.1	10	0.043
Chicago	125	31.7	16	0.068
Cleveland	157	33.6	15	0.063
Denver	89	20.9	12	0.052
Fresno	89	7.4	4.4	0.018
Harrisburg	125	16.6	8.6	0.036
Hartford	126	21.9	11	0.047
Houston	101	16.3	9.3	0.039
Huntington	142	8.2	3.9	0.016
Las Vegas	27	25.9	19	0.079
Lincoln	91	31.1	18	0.077
Little Rock	104	14.4	8.1	0.034
Los Angeles	33	14.7	11	0.044
Miami	128	25.5	13	0.054
Minneapolis	113	35.2	19	0.080
Philadelphia	117	25.6	14	0.057
Phoenix	37	6.8	4.8	0.020
Portland, ME	129	23.0	12	0.049
Raleigh-Durham	110	14.5	8.0	0.033
Salem, OR	146	15.2	7.2	0.030
Salt Lake City	92	20.1	12	0.049
San Francisco	63	37.4	24	0.10
Seattle	157	22.1	9.9	0.041
Winnemucca	67	17.2	11	0.046

5.0 Dispersion Modeling

Dispersion describes the transport of chemical emissions through the air to a receptor. In this risk analysis, dispersion modeling was used to estimate air concentrations associated with a unit emission ($1 \mu\text{g}/\text{m}^2\text{-s}$) (unitized air concentrations, or UACs) at a variety of potential receptor locations. The following sections discuss model selection, the critical parameters of the model, and the model results or UACs.

5.1 Model Selection

A number of dispersion models are available on the EPA Support Center for Regulatory Air Models (SCRAM) Bulletin Board (<http://www.epa.gov/scram001/>). These dispersion models were developed for a variety of applications and each has its own strengths and weaknesses. This analysis required a model with the capability to model dispersion of vapors and particulates from landfills, land application units, wastepiles, and tanks to receptors both on- and offsite for chronic, subchronic, and acute averaging times. Therefore, a dispersion model was needed that could model (1) area sources; (2) ground-level and elevated sources; (3) onsite and offsite impacts; (4) vapors and particulates; and (5) annual, monthly, and daily averaging times.

Five models were considered for this analysis:

- Industrial Source Complex - Short Term v.3 (ISCST3) - U.S. EPA, 1995
- Industrial Source Complex - Long Term v.3 (ISCLT3) - U.S. EPA, 1995
- Toxic Modeling System - Short Term (TOXST) - U.S. EPA, 1994c
- Fugitive Dust Model (FDM) - U.S. EPA, 1992
- COMPDEP - U.S. EPA, 1990.

Table 5-1 summarizes the capabilities of these commonly used air dispersion models with respect to the requirements of this analysis. The ISCST3 (U.S. EPA, 1995) was selected for all aspects of this analysis because it met all the criteria. This model, however, has considerable run times, which limited the number of meteorological stations included in this analysis.

5.2 Critical Parameters

This section discusses the critical parameters of the selected model, ISCST3, and the results of sensitivity analyses performed to investigate several of the model parameters. Results of the sensitivity analyses are presented in Appendix F.

Table 5-1. Air Dispersion Model Capabilities

Model	Source Geometry	Source Height		Receptor Location		Chemical Phase		Averaging Period		
	Area Source	Elevated	Ground	Onsite Air Conc.	Offsite Air Conc.	Vapor	Particulate	Annual	Monthly	Daily
ISCST3	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
ISCLT3	✓	✓	✓	✓	✓	✓	✓	✓		
TOXST	✓	✓	✓		✓	✓	✓	✓	✓	✓
FDM	✓	✓	^a	✓	✓		✓	✓		✓
COMPDEP		✓			✓	✓	✓	✓		✓

^aMinimum height of source for modeling is 0.5 meters.

5.2.1 General Assumptions

This section discusses depletion, rural vs. urban, and terrain assumptions.

5.2.1.1 Depletion. Air concentrations can be calculated in ISCST3 with or without wet and dry depletion. Modeled concentrations without depletions are higher than those with depletions. A sensitivity analysis was conducted that showed that the differences in the maximum concentrations with depletion and without depletion are small at close-to-source receptors, increasing only slightly as the distance from the source increases. The sensitivity analysis also shows that the run time for calculating concentrations using the ISCST3 model with depletion options is 15 to 30 times longer than the run time without depletions for the 5th and 95th percentile of the sizes of land application units. (The difference is greater for larger sources; see sensitivity analysis in Appendix F for details.) Therefore, concentrations were calculated without depletions in this analysis so that a greater number of meteorological locations could be modeled in the time available.

Assumptions Made for Dispersion Modeling

- Dry and wet depletion options were not activated in the dispersion modeling.
- The rural option was used in the dispersion modeling since the types of WMUs being assessed are typically in nonurban areas.
- Flat terrain was assumed.
- An area source was modeled for all WMUs.
- To minimize error due to site orientation, a square area source with sides parallel to X- and Y- axes was modeled.
- Receptor points were placed on 0, 25, 50, 75, 150, 500, and 1,000 m receptor squares starting from the edge of the source with 16 receptor points on each square.
- Modeling was conducted using a unit emission rate of $1 \mu\text{g/s-m}^2$.

5.2.1.2 Rural vs. Urban. ISCST3 may be run in rural or urban mode, depending on land use within a 3-km radius from the source. These modes differ with respect to wind profile exponent and potential temperature gradients. Unless the site is located in a heavily metropolitan

area, the rural option is generally more appropriate. Because the types of WMUs being assessed are typically in nonurban areas, the rural option was used in this analysis.

5.2.1.3 Terrain. Flat terrain for both the source and the surrounding area was assumed in the modeling analysis for two reasons: (1) ISCST3 models all area sources as flat, and (2) complex terrain simulations in the surrounding area result in air concentrations that are highly dependent upon site-specific topography. A specific WMU's location in relation to a hill or valley produces results that would not be applicable to other locations. Complex terrain applications are extremely site-specific; therefore, model calculations from one particular complex terrain location cannot be applied to another. Conversely, simulations from flat terrain produce values that are more universally applicable.

5.2.2 Meteorological Stations and Data

Meteorological data at over 200 meteorological stations in the United States are available on the SCRAM Bulletin Board (<http://www.epa.gov/scram001>) and from a number of other sources. A set of 29 meteorological stations selected in an assessment for EPA's Superfund program Soil Screening Levels (SSLs) (EQM, 1993) as being representative of the nine general climate regions of the continental United States was used in this analysis.

In EPA's SSL study, it was determined that 29 meteorological stations would be a sufficient sample to represent the population of 200 meteorological stations and predict mean dispersion values with a high (95 percent) degree of confidence. The 29 meteorological stations were distributed among nine climate regions based on meteorological representativeness and variability across each region.

These climate regions were:

- North Pacific Coastal
- Northwest Mountains
- Midwest
- South Pacific Coastal
- Central Plains
- Northern Atlantic
- Southwest
- Southeast
- South Florida.

Large-scale regional average conditions were used to select the actual stations (EQM, 1993).

The 29 meteorological stations are listed in Table 5-2. To assign each Industrial D facility to a meteorological station, EPA used a Geographic Information System (GIS) to construct Thiessen polygons around each station that enclose the areas closest to each station. The boundaries of these areas were then adjusted to ensure that each boundary encloses an area that is most similar in meteorological conditions to those measured at the meteorological station. To assist in this process, a GIS coverage of Bailey's ecoregion divisions and provinces (Bailey et al., 1994) was used to conflate the boundaries to correspond to physiographic features likely to influence climate or boundaries corresponding to changes in temperature or precipitation. General wind regimes were also considered in the conflation process.

Key factors considered in the conflation process include: defining coastal regimes as narrow polygons, which generally stretched about 25 to 50 miles inland, to capture regions

Table 5-2. Meteorological Stations Used in the Air Characteristic Study

City	Met Station		Latitude		Longitude	
	State	#	Degree	Minute	Degree	Minute
Albuquerque	NM	23050	35	3	106	37
Atlanta	GA	13874	33	39	84	25
Bismarck	ND	24011	46	46	100	45
Boise	ID	24131	43	34	116	13
Casper	WY	24089	42	55	106	28
Charleston	SC	13880	32	54	80	2
Chicago	IL	94846	41	59	87	54
Cleveland	OH	14820	41	25	81	52
Denver	CO	23062	39	46	104	52
Fresno	CA	93193	36	46	119	43
Harrisburg	PA	14751	40	13	76	51
Hartford	CT	14740	41	56	72	41
Houston	TX	12960	29	58	95	21
Huntington	WV	03860	38	22	82	33
Las Vegas	NV	23169	36	5	115	10
Lincoln	NE	14939	40	51	96	45
Little Rock	AR	13963	34	44	92	14
Los Angeles	CA	23174	33	56	118	24
Miami	FL	12839	25	49	80	17
Minneapolis	MN	14922	44	53	93	13
Philadelphia	PA	13739	39	53	75	15
Phoenix	AZ	23183	33	26	112	1
Portland	ME	14764	43	39	70	19
Raleigh-Durham	NC	13722	35	52	78	47
Salem	OR	24232	44	55	123	0
Salt Lake City	UT	24127	40	47	111	57
San Francisco	CA	23234	37	37	122	23
Seattle	WA	24233	47	27	122	18
Winnemucca	NV	24128	40	54	117	48

Source: EQM (1993).

dominated by coastal climate effects; maintaining tropical/subtropical and arid/semiarid divisions in the southwestern United States; and using the ecoregion boundaries in Washington, Oregon, and California to separate the more humid marine/redwood or Mediterranean mountain regimes from the deserts to the east. In general, Thiessen polygons were used to define the meteorological station areas for the remainder of the country.

Based on facility locations derived from Industrial D survey data, the Industrial D sites were then overlaid on the GIS coverage of the conflated meteorological boundaries and meteorological station assignments were then exported for use in the modeling exercise. Four sites in Alaska and four in Hawaii were deleted from the analysis at this point because the 29 meteorological stations are limited to the continental United States. Figure 5-1 shows the final

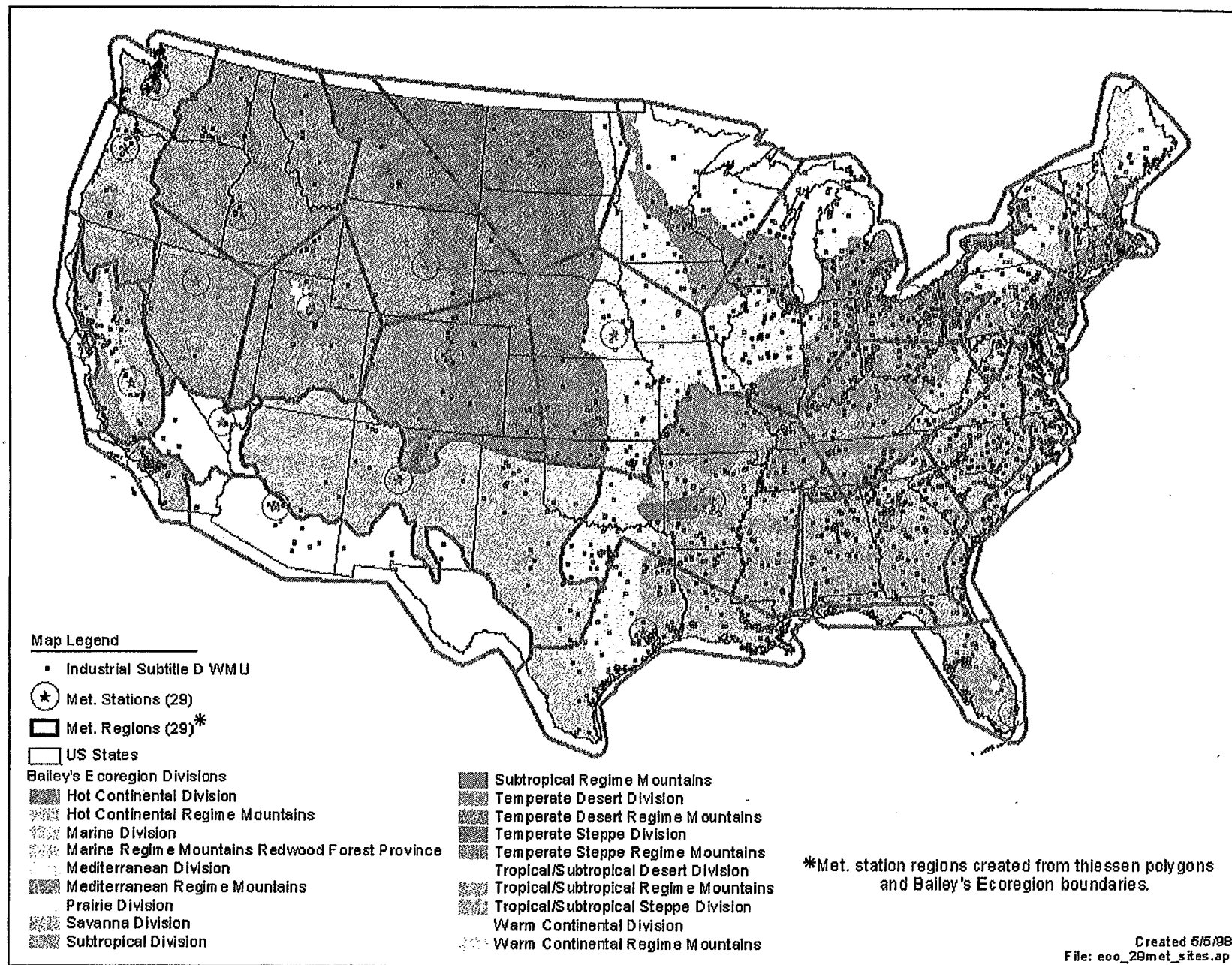


Figure 5-1. Meteorological station regions.

meteorological station boundaries used for the study along with the locations of the Industrial D facility sites.

The modeling analysis was conducted using 5 years of representative meteorological data from each of the 29 meteorological stations. Five-year wind roses representing the frequency of wind directions and windspeeds for the 29 meteorological stations were analyzed. These show that the 29 meteorological stations represent a variety of wind patterns and are presented in Appendix F.

**Shape of Wind Rose for
29 Meteorological Stations**

Shape of Wind Rose	No. of Stations
Narrowly distributed	10
Moderately distributed	4
Evenly distributed	6
Bimodally distributed	9

**Meteorological Data for
the ISCST3 Model
without Depletion**

Wind Direction (or Flow Vector)
Windspeed
Ambient Temperature
Stability Class
Mixing Height

Wind direction and windspeed are typically the most important meteorological inputs for dispersion modeling analysis. Wind direction determines the direction of the greatest impacts. Windspeed is inversely proportional to ground-level air concentrations, so that the lower the windspeed, the higher the air concentration.

Mixing height determines the heights to which pollutants can be diffused vertically. Stability class is also an important factor in determining the rate of lateral

and vertical diffusion. The more unstable the air, the greater the diffusion. This increase would lower centerline concentration.

5.2.3 Source Release Parameters

This section describes the source parameters and assumptions used in the dispersion modeling, including source type and elevation, source shape and orientation, and source areas.

5.2.3.1 Source Type and Elevation. All WMU types modeled in this analysis were modeled as area sources. Landfills and land application units were modeled as ground-level sources, and wastepiles and tanks were modeled as elevated sources.

5.2.3.2 Source Shape and Orientation. The ISCST3 models an area source as a rectangle or combination of rectangles. The user may also specify an angle of rotation relative to a north-south orientation. A sensitivity analysis was conducted to compare the air concentrations from a square area source, a rectangular area source oriented east to west, and a rectangular area source oriented north to south to determine what role source shape and orientation play in determining dispersion coefficients of air pollutants. The results show that the differences in unitized air concentration between the square area source and the two rectangular area sources are less than the differences between the two rectangular sources. In addition, a square area source has the least amount of impact on orientation. Because information on source shapes or

orientations is not available, a square source was chosen to minimize the errors caused by source shapes and orientations. (See sensitivity analysis in Appendix F for details.)

5.2.3.3 Source Areas Modeled. In the modeling analysis, five types of WMUs were considered (i.e., landfill, land application unit, wastepile, aerated tank, and storage tank). Because the ISCST3 model is sensitive to the size of the area source, the relationship between air concentrations and size of the area source was analyzed. As illustrated in Figure 5-2, the results show that, for relatively small area sources, air concentrations increase significantly as the size of the area source increases. For large area sources, this increase in air concentrations is not as significant.

In order to address this model sensitivity, yet avoid modeling approximately 2,000 separate WMUs, EPA developed area strata that represented the distribution of the surface area for each of the WMU types. Landfills and land application units were modeled as ground-level area sources, while wastepiles and tanks were treated as elevated area sources. Separate area strata were developed for ground-level and elevated sources. In addition, separate areas were modeled for tanks, because these were based on model units, rather than the Industrial D Survey database. (See Section 3 for further details.) Fourteen area strata were selected for landfills and land application units, seven for wastepiles, and two for tanks. The median area size for each stratum was used in the dispersion modeling analysis. Tables 5-3, 5-4, and 5-5 present the source areas and heights used in the modeling analysis.

This provided a set of UACs for use in the analysis. For any specific WMU, a UAC was then estimated using an interpolation routine that used the UACs associated with modeled areas immediately above and below the actual area of the unit. The interpolation routine provides a technique for minimizing the number of ISCST3 runs required for a WMU while also minimizing the error associated with the difference between the UACs for preselected areas and the UAC for the actual area of the WMU. The interpolation is described in more detail in Section 7.

5.2.4 Receptors

The ISCST3 model allows the user to specify receptors with Cartesian receptor grid and/or polar receptor grid. In general, Cartesian receptors are used for near-source receptors and polar grid receptors for more distant receptors. The number of receptors modeled greatly impacts run time. However, if too few receptors are modeled, the location of peak concentration may be missed. A sensitivity analysis was conducted to determine receptor locations and spacings that would provide adequate resolution without modeling an excessive number of receptors. (See Appendix F for details.) The results of the sensitivity analysis show that the maximum concentrations are generally higher for a dense receptor grid (i.e., 64 or 32 receptors on each square) than for a scattered receptor grid (i.e., 16 receptors on each square). However, the differences of the maximum receptor concentrations are not significant between a dense and a scattered receptor grid. Therefore, 16 evenly spaced receptor points on each square were used in the modeling. The sensitivity analysis also shows that the maximum downwind concentrations decrease sharply from the edge of the area source to about 1,000 meters from the source. After the first 1,000 meters from the edge of the area source, concentrations decrease very slowly as the

downwind distance increases. Therefore, for annual average concentrations, the receptor points were placed on 0, 25, 50, 75, 150, 500, and 1,000 meter receptor squares starting from the edge of the source, with 16 receptor points on each square. The first receptor square (i.e., 0 meter) is at the edge of the unit. For monthly and daily averaging periods used in the subchronic and acute assessment, the receptors were placed on 0, 25, 50, and 75 meter receptor squares.

5.3 Unitized Air Concentrations

Unitized air concentrations (UACs) were calculated by running ISCST3 with a unit emission rate (i.e., $1 \mu\text{g}/\text{m}^2\text{-s}$). The selected areas for each type of WMU were modeled with 29 representative meteorological locations in the continental United States to estimate UACs. The 5-year average UACs at all receptor points were calculated for the long-term or chronic exposure scenario. They were used as inputs to the Monte Carlo analysis and as input to the interpolation routine discussed above.

A similar methodology and assumptions were used to model dispersion for acute and subchronic exposures. Since the ISCST3 model uses hourly meteorological data, the outputs from the model can be used to develop any averaging times equal to or greater than 1 hour. One set of ISCST3 runs (for the 21 areas and 29 meteorological stations) was done for both acute and subchronic, resulting in 5 years of hourly average concentrations at each receptor. For each area, meteorological location, and receptor location, the maximum air concentration for any 24-hour period over the 5 years was selected. Then, for each area and meteorological station, the maximum 24-hour air concentration among all receptor locations at each distance modeled was selected, and this was used as the UAC for that area and meteorological station for acute exposure. The same method was used to determine the subchronic UAC, except that the maximum 30-day period over the 5 years was used instead of the maximum 24-hour period. It was assumed that the greatest risk of acute exposure would be closest to the site; therefore, the receptors points were placed at 0, 25, 50, and 75 meters from the edge of the WMU, with 16 equally spaced directions at each distance.

The maximum annual average UACs are presented in Tables 5-6 through 5-8 for the different types of WMUs. Typically, the location of maximum impacts with respect to the source are determined by the prevailing wind direction. For ground-level area sources (i.e., landfills and land application units), maximum annual average UACs are always located on the first receptor square (i.e., 0-m receptors or onsite receptors). For elevated area sources, the maximum annual average UACs are usually located on the first receptor square and occasionally located on the second or third receptor square. The results in Tables 5-6 through 5-8 show that the annual average UACs increase with the increasing area size of the sources.

Figures 5-3 through 5-6 show that maximum UACs vary with meteorological location. For landfills and land application units, the maximum UACs at some meteorological locations can be twice as much as those at other locations. For wastepiles and tanks, the maximum UACs at some meteorological locations are more than twice those at other meteorological locations.

Table 5-6. Maximum Annual Average Unitized Air Concentrations ($\mu\text{g}/\text{m}^3$ / $\mu\text{g}/\text{s}\cdot\text{m}^2$) for Landfills and Land Application Units

Met Station	Station No.	Area (m^2)													
		81	567	1,551	4,047	12,546	40,500	78,957	161,880	243,000	376,776	607,000	906,529	1,408,356	8,090,000
Albuquerque, NM	23050	3.521	5.791	7.103	8.450	10.175	12.112	13.316	14.535	15.487	16.406	17.299	18.206	19.287	25.002
Atlanta, GA	13874	3.919	6.369	7.789	9.236	11.119	13.224	14.526	15.927	16.902	17.896	18.937	19.950	21.142	27.323
Bismarck, ND	24011	3.598	5.871	7.182	8.528	10.273	12.231	13.443	14.816	15.650	16.579	17.620	18.566	19.667	25.220
Boise, ID	24131	4.806	7.739	9.458	11.251	13.543	16.138	17.770	19.508	20.710	21.978	23.311	24.550	26.052	33.867
Casper, WY	24089	3.532	5.718	6.980	8.265	9.923	11.790	12.931	14.184	15.020	15.892	16.833	17.724	18.751	24.085
Charleston, SC	13880	3.760	6.134	7.503	8.907	10.733	12.778	14.045	15.392	16.350	17.320	18.316	19.302	20.451	26.415
Chicago, IL	94846	3.678	6.011	7.356	8.726	10.505	12.493	13.712	14.980	15.944	16.871	17.797	18.741	19.843	25.626
Cleveland, OH	14820	4.163	6.639	8.064	9.519	11.415	13.527	14.833	16.268	17.227	18.232	19.308	20.341	21.564	27.959
Denver, CO	23062	5.364	8.645	10.541	12.488	15.039	17.898	19.690	21.634	22.945	24.336	25.798	27.217	28.886	37.541
Fresno, CA	93193	5.783	9.460	11.587	13.794	16.611	19.800	21.792	24.024	25.383	26.916	28.634	30.144	31.955	41.022
Harrisburg, PA	14751	4.291	6.892	8.380	9.900	11.877	14.073	15.434	16.882	17.900	18.937	20.006	21.060	22.298	28.745
Hartford, CT	14740	4.478	7.454	9.176	10.934	13.216	15.775	17.344	18.848	20.221	21.412	22.470	23.684	25.101	32.702
Houston, TX	12960	4.137	6.811	8.352	9.925	11.961	14.239	15.632	17.227	18.189	19.244	20.448	21.531	22.784	28.985
Huntington, WV	3860	5.548	9.154	11.240	13.378	16.161	19.282	21.207	23.265	24.728	26.197	27.720	29.218	30.966	39.932
Las Vegas, NV	23169	4.353	7.072	8.645	10.254	12.349	14.700	16.159	17.697	18.816	19.941	21.081	22.222	23.557	30.668
Lincoln, NE	14939	3.007	4.867	5.936	7.027	8.445	10.027	11.000	12.036	12.781	13.525	14.291	15.051	15.939	20.577
Little Rock, AR	13963	4.500	7.402	9.079	10.795	13.023	15.528	17.065	18.732	19.883	21.053	22.296	23.486	24.888	32.110
Los Angeles, CA	24174	4.492	7.480	9.269	11.100	13.457	16.112	17.745	19.332	20.709	21.944	23.083	24.311	25.753	33.445
Miami, FL	12839	3.752	6.150	7.550	8.984	10.845	12.944	14.240	15.718	16.612	17.608	18.731	19.750	20.932	26.829
Minneapolis, MN	14922	3.334	5.453	6.676	7.924	9.541	11.354	12.464	13.676	14.502	15.347	16.253	17.121	18.127	23.300
Philadelphia, PA	13739	4.359	7.076	8.643	10.243	12.317	14.644	16.076	17.596	18.689	19.784	20.908	22.021	23.317	30.083
Phoenix, AZ	23183	5.640	9.043	11.002	13.016	15.650	18.591	20.439	22.494	23.763	25.185	26.729	28.164	29.850	30.083
Portland, ME	14764	5.028	8.269	10.146	12.070	14.574	17.389	19.127	20.946	22.310	23.642	24.983	26.344	27.933	36.239
Raleigh-Durham, NC	13722	4.407	7.196	8.805	10.453	12.599	14.999	16.483	18.079	19.192	20.327	21.510	22.665	24.018	30.956
Salem, OR	24232	4.580	7.348	8.939	10.567	12.687	15.053	18.120	18.120	19.185	20.308	21.513	22.661	24.005	31.007
Salt Lake City, UT	24127	4.735	7.576	9.218	10.909	13.095	15.546	18.754	18.754	19.865	21.050	22.318	23.521	24.956	32.412
San Francisco, CA	23234	4.500	7.257	8.842	10.465	12.585	14.946	17.977	17.977	19.084	20.213	21.376	22.524	23.882	30.988
Seattle, WA	24233	4.276	6.799	8.231	9.691	11.592	13.686	16.390	16.390	17.324	18.310	19.359	20.365	21.547	27.722
Winnemucca, NV	24128	4.123	6.720	8.222	9.763	11.772	14.028	16.889	16.889	17.980	19.055	20.130	21.224	22.505	29.215

Table 5-7. Maximum Annual Average Unitized Air Concentrations ($\mu\text{g}/\text{m}^3$ / $\mu\text{g}/\text{s}\cdot\text{m}^2$) for Wastepiles

Met Station	Station No.	Area (m^2) (2-m Height Wastepiles)							Area Strata (m^2) (5-m Height Wastepiles)						
		20	162	486	2,100	10,100	101,000	1,300,000	20	162	486	2,100	10,100	101,000	1,300,000
Albuquerque, NM	23050	0.037	0.171	0.378	0.993	2.359	5.704	11.011	0.014	0.053	0.107	0.288	0.824	2.956	7.671
Atlanta, GA	13874	0.043	0.195	0.431	1.141	2.644	6.284	12.066	0.016	0.060	0.120	0.325	0.940	3.312	8.467
Bismarck, ND	24011	0.035	0.155	0.343	0.932	2.273	5.685	11.093	0.013	0.049	0.097	0.258	0.759	2.867	7.693
Boise, ID	24131	0.056	0.235	0.520	1.389	3.183	7.621	14.732	0.021	0.072	0.143	0.384	1.132	3.996	10.383
Casper, WY	24089	0.040	0.181	0.405	1.084	2.461	5.714	10.846	0.015	0.056	0.110	0.301	0.894	3.080	7.678
Charleston, SC	13880	0.038	0.168	0.372	1.003	2.393	5.944	11.581	0.014	0.053	0.105	0.280	0.820	3.008	8.027
Chicago, IL	94846	0.038	0.170	0.380	1.030	2.431	5.897	11.340	0.014	0.053	0.106	0.285	0.845	3.049	7.929
Cleveland, OH	14820	0.049	0.214	0.479	1.251	2.897	6.712	12.611	0.018	0.064	0.128	0.353	1.038	3.634	9.059
Denver, CO	23062	0.054	0.237	0.518	1.401	3.393	8.397	16.369	0.020	0.075	0.148	0.391	1.137	4.262	11.383
Fresno, CA	93193	0.077	0.344	0.744	1.858	4.018	9.168	17.785	0.028	0.101	0.205	0.562	1.556	5.002	12.248
Harrisburg, PA	14751	0.047	0.214	0.477	1.269	2.978	6.960	13.027	0.018	0.066	0.131	0.357	1.049	3.731	9.318
Hartford, CT	14740	0.049	0.212	0.474	1.283	2.999	7.096	14.060	0.018	0.067	0.132	0.354	1.050	3.762	9.585
Houston, TX	12960	0.042	0.191	0.424	1.129	2.696	6.640	12.839	0.016	0.059	0.119	0.320	0.933	3.392	8.910
Huntington, WV	3860	0.057	0.248	0.548	1.450	3.416	8.647	17.196	0.021	0.077	0.153	0.410	1.191	4.284	11.707
Las Vegas, NV	23169	0.045	0.194	0.432	1.185	2.852	6.949	13.504	0.017	0.062	0.122	0.323	0.961	3.588	9.440
Lincoln, NE	14939	0.032	0.142	0.317	0.867	2.046	4.850	9.212	0.012	0.045	0.088	0.237	0.708	2.566	6.520
Little Rock, AR	13963	0.045	0.201	0.442	1.181	2.830	7.049	13.894	0.017	0.063	0.126	0.335	0.967	3.553	9.533
Los Angeles, CA	24174	0.055	0.255	0.564	1.466	3.232	7.230	14.069	0.020	0.076	0.153	0.465	1.263	4.022	9.655
Miami, FL	12839	0.041	0.181	0.404	1.080	2.521	6.016	11.650	0.015	0.056	0.112	0.303	0.889	3.163	8.083
Minneapolis, MN	14922	0.033	0.147	0.326	0.896	2.168	5.320	10.290	0.013	0.047	0.093	0.246	0.729	2.726	7.166
Philadelphia, PA	13739	0.045	0.198	0.439	1.200	2.876	6.962	13.365	0.017	0.063	0.124	0.330	0.978	3.610	9.369
Phoenix, AZ	23183	0.062	0.274	0.597	1.555	3.628	8.793	16.962	0.023	0.085	0.170	0.455	1.281	4.533	11.828
Portland, ME	14764	0.046	0.196	0.433	1.209	3.056	7.866	15.636	0.018	0.065	0.126	0.327	0.972	3.857	10.701
Raleigh-Durham, NC	13722	0.043	0.191	0.424	1.152	2.802	6.956	13.566	0.016	0.061	0.120	0.320	0.936	3.523	9.394
Salem, OR	24232	0.048	0.209	0.466	1.287	3.060	7.288	13.859	0.018	0.067	0.130	0.347	1.045	3.844	9.833
Salt Lake City, UT	24127	0.052	0.232	0.514	1.386	3.218	7.569	14.453	0.020	0.072	0.142	0.383	1.131	4.041	10.268
San Francisco, CA	23234	0.046	0.207	0.464	1.252	2.975	7.163	13.747	0.018	0.065	0.127	0.345	1.029	3.743	9.704
Seattle, WA	24233	0.053	0.240	0.540	1.440	3.187	7.022	12.804	0.020	0.073	0.145	0.399	1.193	3.974	9.363
Winnemucca, NV	24128	0.040	0.172	0.380	1.040	2.555	6.432	12.676	0.015	0.056	0.109	0.287	0.842	3.211	8.724

Table 5-8. Maximum Annual Average Unitized Air Concentrations ($\mu\text{g}/\text{m}^3/\mu\text{g}/\text{s}\cdot\text{m}^2$) for Aerated and Storage Tanks

Met Station	Station	Area (m^2)	
	No.	27	430
Albuquerque, NM	23050	0.00286	0.04652
Atlanta, GA	13874	0.00333	0.06414
Bismarck, ND	24011	0.00245	0.04142
Boise, ID	24131	0.00519	0.09329
Casper, WY	24089	0.00425	0.08087
Charleston, SC	13880	0.00257	0.04466
Chicago, IL	94846	0.00248	0.04656
Cleveland, OH	14820	0.00408	0.07670
Denver, CO	23062	0.00383	0.06834
Fresno, CA	93193	0.00652	0.12357
Harrisburg, PA	14751	0.00378	0.06610
Hartford, CT	14740	0.00462	0.07620
Houston, TX	12960	0.00321	0.06281
Huntington, WV	3860	0.00403	0.07845
Las Vegas, NV	23169	0.00265	0.04930
Lincoln, NE	14939	0.00336	0.05724
Little Rock, AR	13963	0.00272	0.04850
Los Angeles, CA	24174	0.00779	0.12923
Miami, FL	12839	0.00328	0.05823
Minneapolis, MN	14922	0.00235	0.04401
Philadelphia, PA	13739	0.00350	0.05938
Phoenix, AZ	23183	0.00506	0.08872
Portland, ME	14764	0.00317	0.05184
Raleigh-Durham, NC	13722	0.00302	0.05285
Salem, OR	24232	0.00532	0.08962
Salt Lake City, UT	24127	0.00465	0.08360
San Francisco, CA	23234	0.00543	0.09108
Seattle, WA	24233	0.00594	0.10704
Winnemucca, NV	24128	0.00282	0.04978

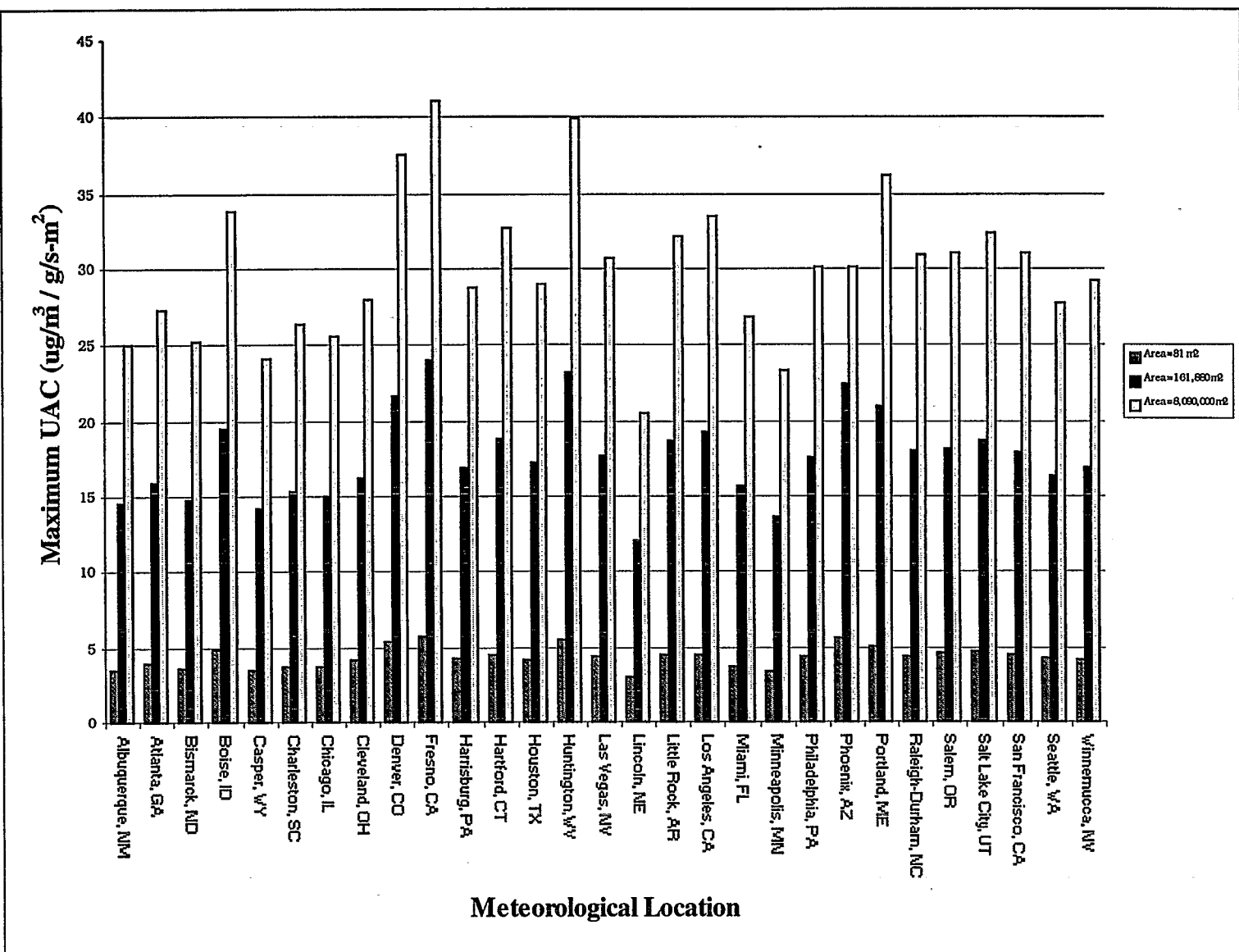


Figure 5-3. Maximum UAC by meteorological location (landfills and LAUs).

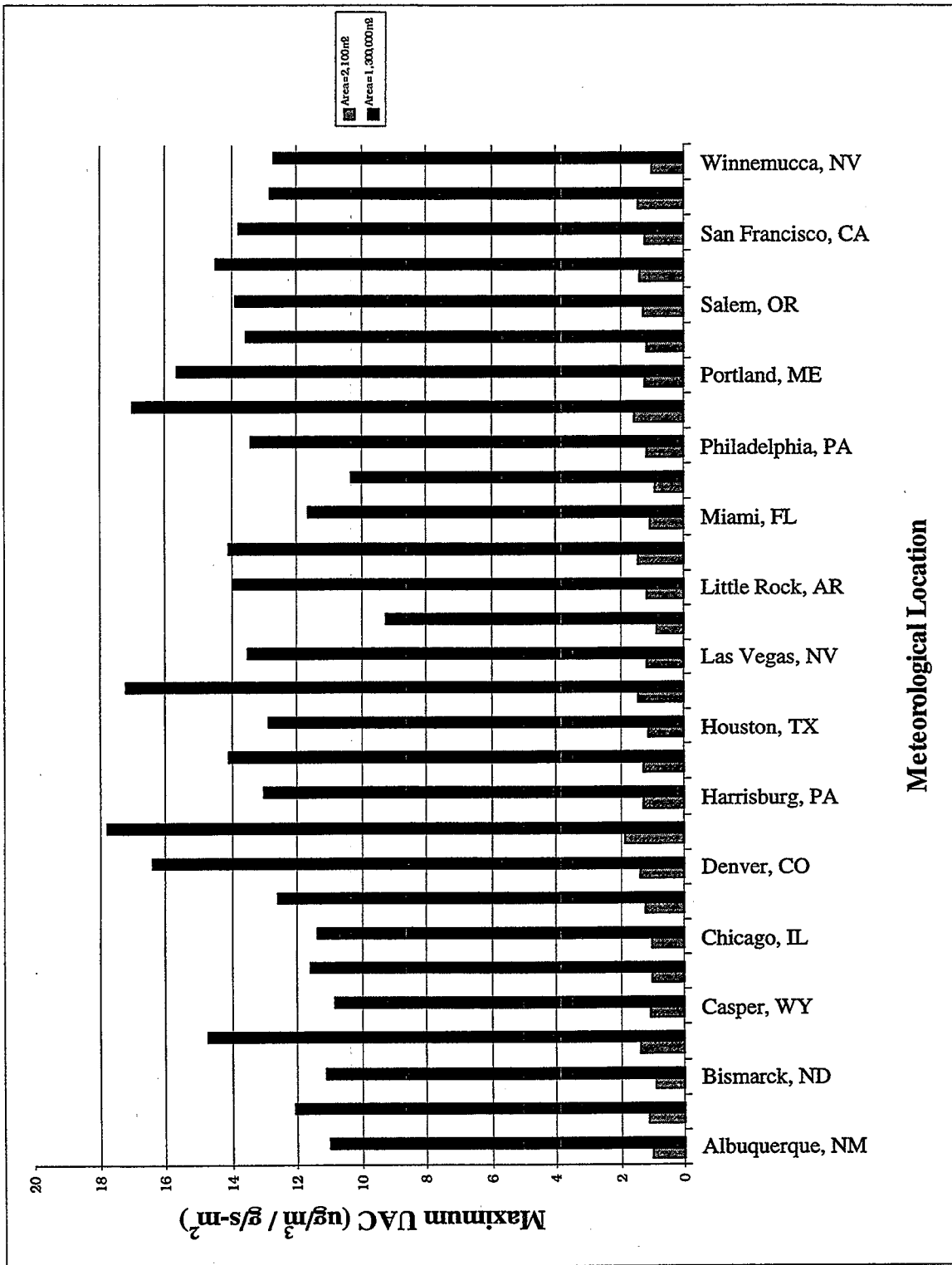


Figure 5-4. Maximum UAC by meteorological location (2-m wastepiles).

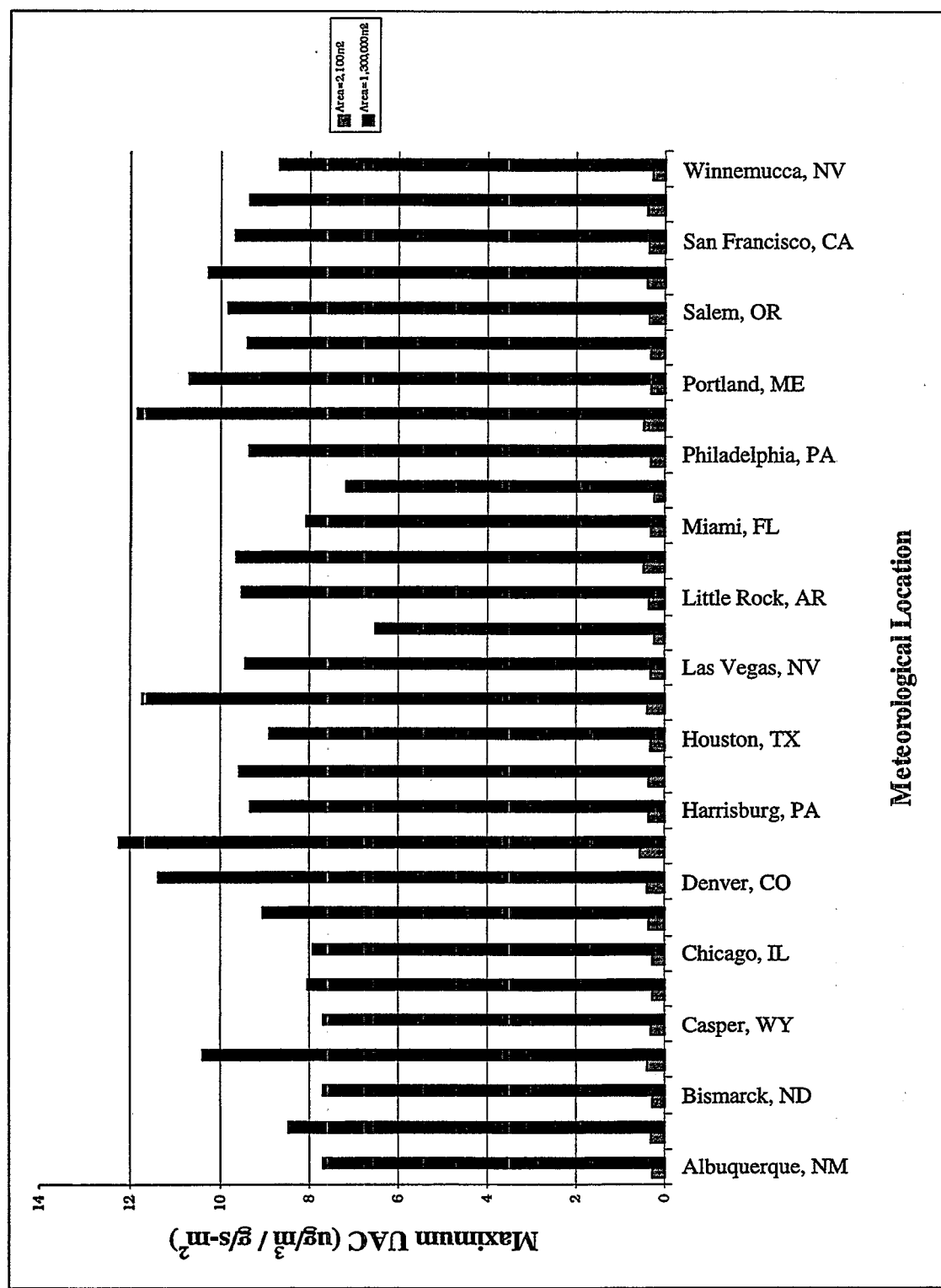


Figure 5-5. Maximum UAC by meteorological location (5-m wastepiles).

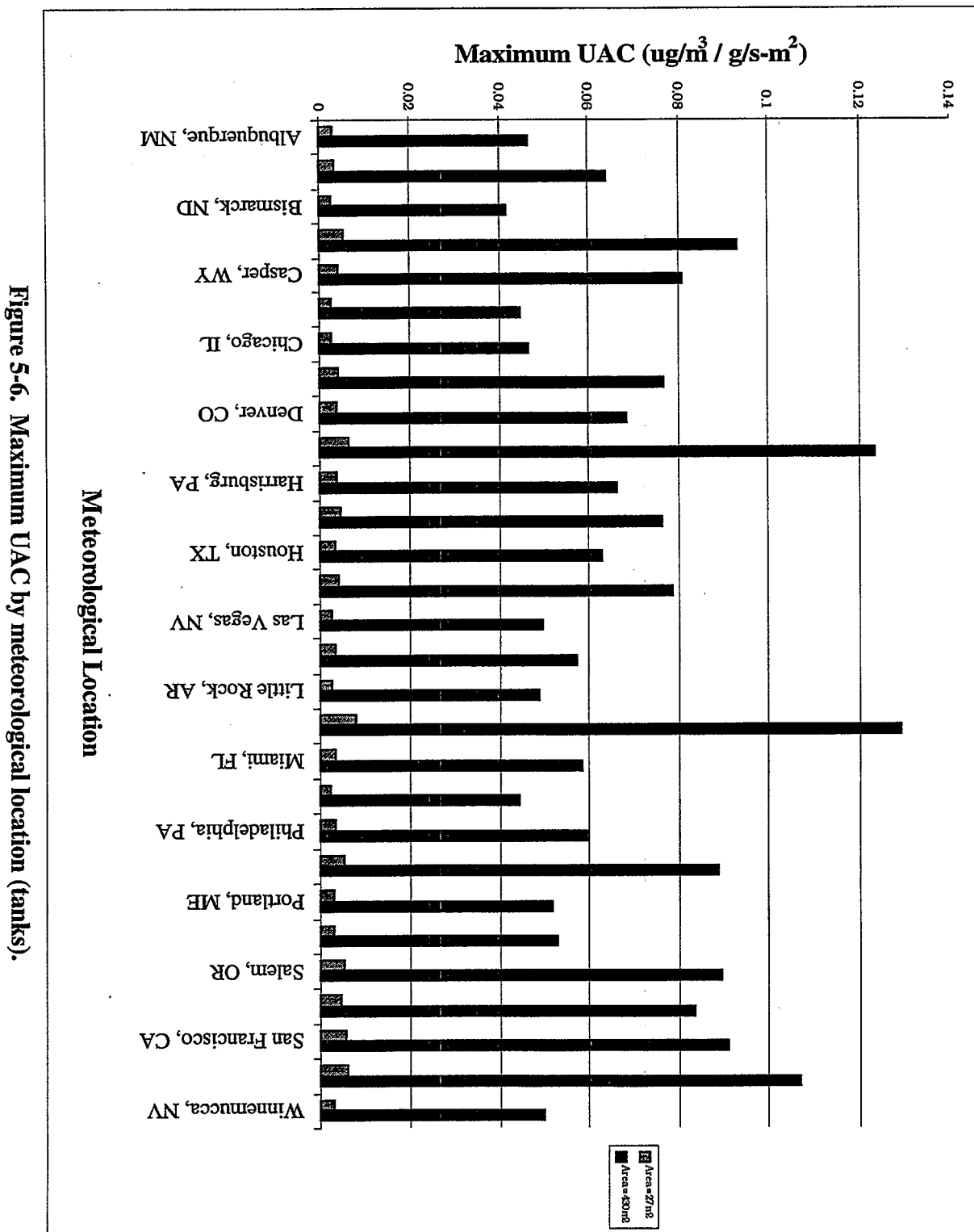
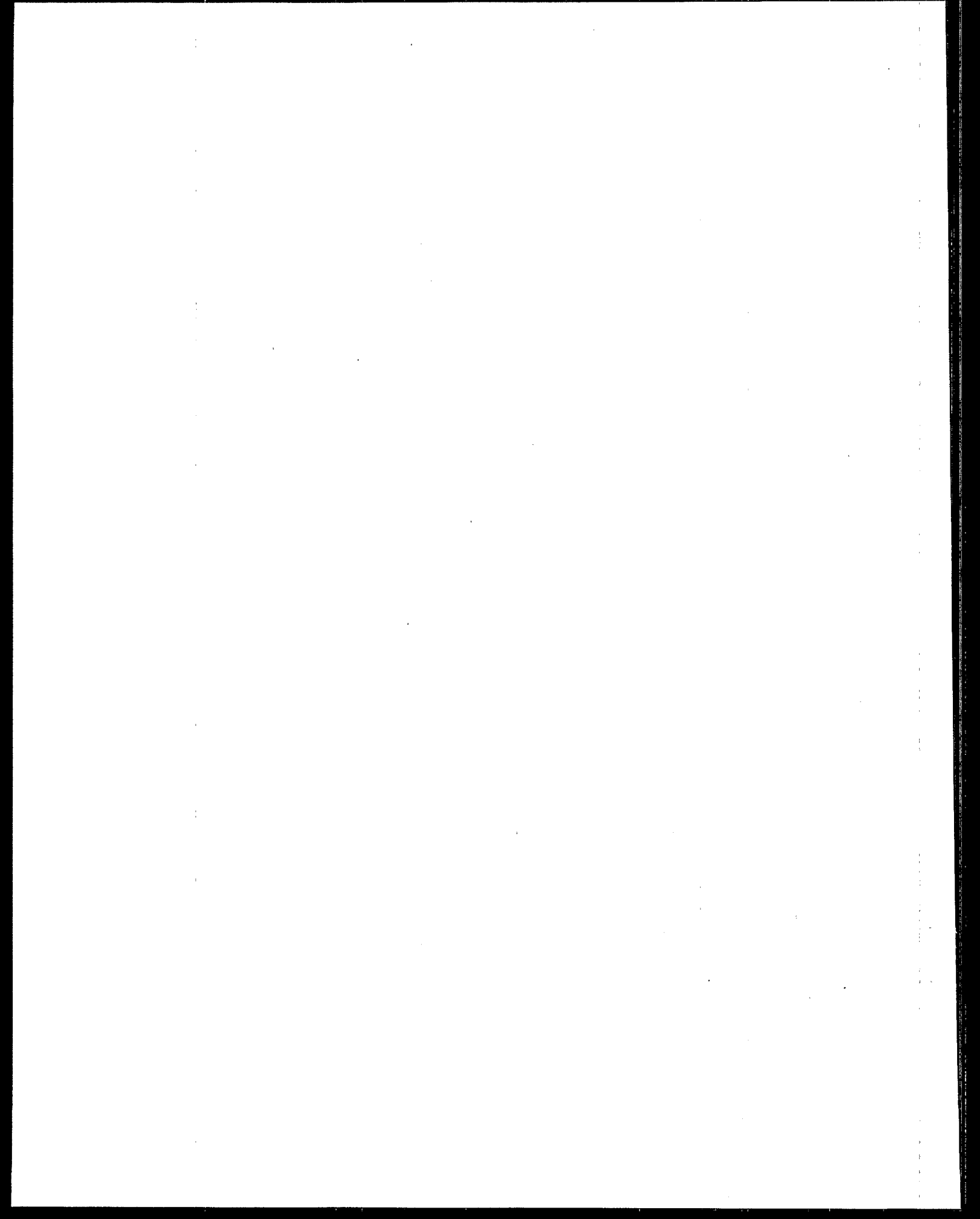


Figure 5-6. Maximum UAC by meteorological location (tanks).



6.0 Development of Inhalation Health Benchmarks

Inhalation health benchmarks for chronic, subchronic, and acute exposure durations were needed. This section describes the health benchmarks used in this study.

6.1 Chronic Inhalation Health Benchmarks Used in This Study

Chronic inhalation health benchmarks used in this study include inhalation reference concentrations (RfCs) for noncarcinogens and inhalation unit risk factors (URFs) and inhalation cancer slope factors (CSFs) for carcinogens. URFs and CSFs were used in the analysis for carcinogenic constituents, regardless of the availability of an RfC. Inhalation health benchmarks were identified in the Integrated Risk Information System (IRIS) and the Health Effects Assessment Summary Tables (HEAST) (U.S. EPA, 1997a, 1998). IRIS and HEAST are maintained by the Agency, and values from IRIS and HEAST were used in the analysis whenever available. Provisional EPA benchmarks and Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels (MRLs) were used to fill in data gaps (see Section 6.1.1). Additional chronic inhalation health benchmarks were derived for use in this analysis for constituents lacking EPA or ATSDR values (see Section 6.1.2).

Figure 6-1 describes the approach used to develop the chronic inhalation health benchmarks used in this analysis. The benchmarks are summarized in Table 6-1.

6.1.1 Alternate Chronic Inhalation Health Benchmarks Identified

If no IRIS or HEAST chronic inhalation health benchmarks were available, benchmarks from alternative sources were sought. Provisional EPA benchmarks, ATSDR inhalation MRLs, and California EPA noncancer chronic reference exposure levels (CalEPA, 1997a) were included whenever available. Alternate RfCs were identified for

- Acetone
- Cyclohexanol
- Isophorone
- Naphthalene
- Phenol
- Pyridine
- Tetrachloroethylene
- 1,1,1-Trichloroethane
- Xylenes.

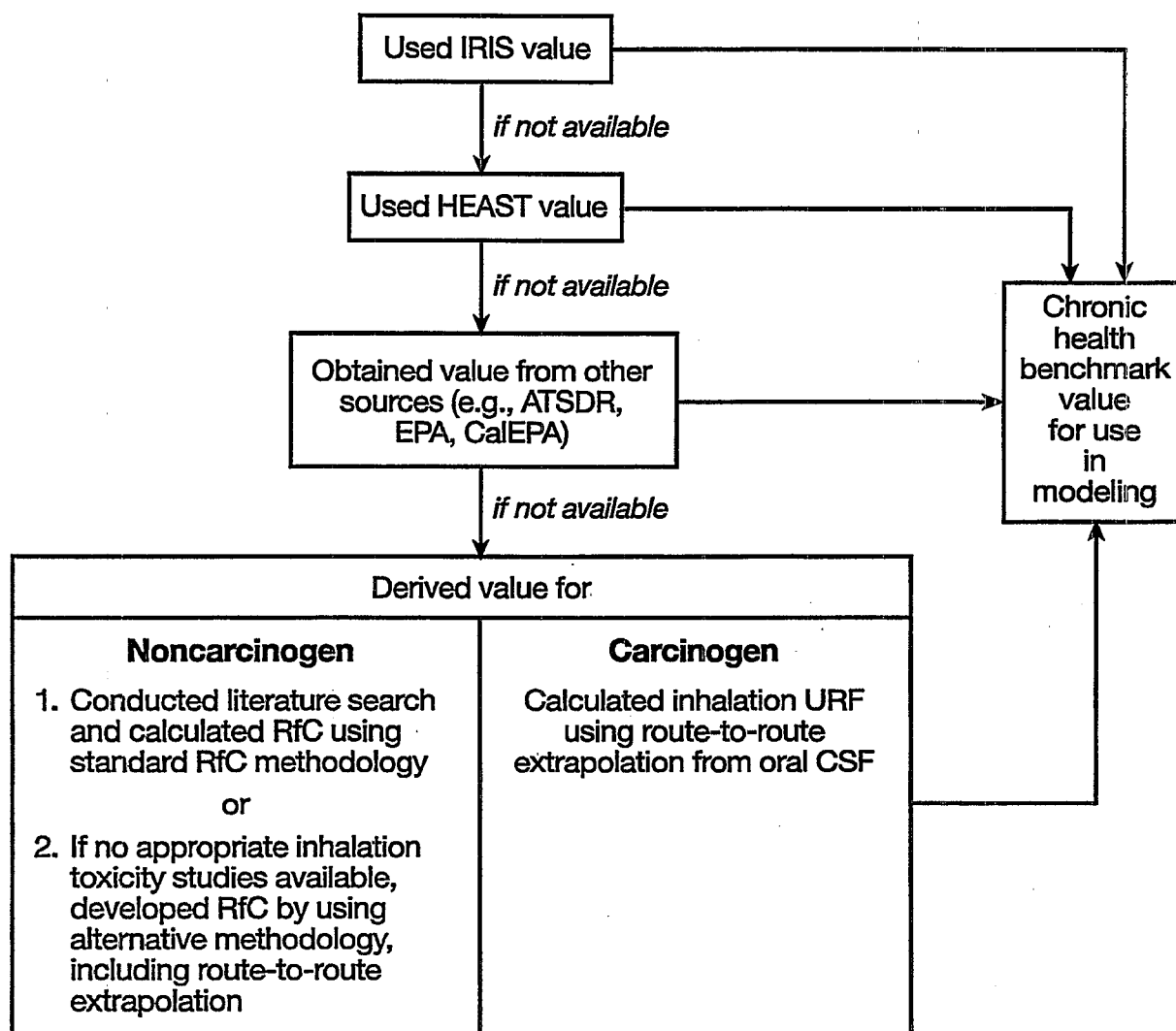


Figure 6-1. Approach used to select chronic inhalation health benchmark values.

**Table 6-1. Chronic Inhalation Health Benchmarks Used in the
Air Characteristic Analysis**

CAS#	Name	Noncarcinogens			Carcinogens		
		RfC (mg/m ³)	RfC Target Organ	Ref ^a	Inhal URF (µg/m ³) ⁻¹	Inhal CSF (mg/kg/d) ⁻¹	Ref ^a
75-07-0	Acetaldehyde	9.0E-03	Respiratory	I	2.2E-06	7.7E-03	I
67-64-1	Acetone	3.1E+01	Neurological	A	NA	NA	
75-05-8	Acetonitrile	5.0E-02	Liver	H	NA	NA	
107-02-8	Acrolein	2.0E-05	Respiratory	I	NA	NA	
79-06-1	Acrylamide	NA			1.3E-03	4.6E+00	I
79-10-7	Acrylic acid	1.0E-03	Respiratory	I	NA	NA	
107-13-1	Acrylonitrile	2.0E-03	Respiratory	I	6.8E-05	2.4E-01	I
107-05-1	Allyl chloride	1.0E-03	Neurological	I	NA	NA	
62-53-3	Aniline	1.0E-03	Spleen	I	NA	NA	
7440-38-2	Arsenic	NA			4.3E-03	1.5E+01	I
7440-39-3	Barium	5.0E-04	Reproductive	H	NA	NA	
71-43-2	Benzene	NA			8.3E-06	2.9E-02	I
92-87-5	Benzidine	NA			6.7E-02	2.3E+02	I
50-32-8	Benzo(a)pyrene	NA			1.7E-03	6.0E+00	
7440-41-7	Beryllium	2.0E-05	Respiratory	I	2.4E-03	8.4E+00	I
75-27-4	Bromodichloromethane	NA			1.8E-05	6.2E-02	D
75-25-2	Bromoform (Tribromomethane)	NA			1.1E-06	3.9E-03	I
106-99-0	Butadiene, 1,3-	NA			2.8E-04	9.8E-01	I
7440-43-9	Cadmium	NA			1.8E-03	6.3E+00	I
75-15-0	Carbon disulfide	7.0E-01	Reproductive	I	NA	NA	
56-23-5	Carbon tetrachloride	NA			1.5E-05	5.3E-02	I
126-99-8	Chloro-1,3-butadiene, 2- (Chloroprene)	7.0E-03	Respiratory	H	NA	NA	
108-90-7	Chlorobenzene	2.0E-02	Kidney and liver	H	NA	NA	
124-48-1	Chlorodibromomethane	NA			2.4E-05	8.4E-02	D
67-66-3	Chloroform	NA			2.3E-05	8.1E-02	I
95-57-8	Chlorophenol, 2-	1.4E-03	Repro/developmental	D	NA	NA	
7440-47-3	Chromium VI	NA			1.2E-02	4.2E+01	I
7440-48-4	Cobalt	1.0E-05	Respiratory	D	NA	NA	
1319-77-3	Cresols (total)	4.0E-04	Hematological	D	NA	NA	
98-82-8	Cumene	4.0E-01	Kidney and adrenal	I	NA	NA	
108-93-0	Cyclohexanol	6.0E-05	Muscle	FR	NA	NA	

(continued)

Table 6-1. (continued)

CAS#	Name	Noncarcinogens			Carcinogens		
		RfC (mg/m ³)	RfC Target Organ	Ref ^a	Inhal URF (µg/m ³) ⁻¹	Inhal CSF (mg/kg/d) ⁻¹	Ref ^a
96-12-8	Dibromo-3-chloropropane, 1,2-	2.0E-04	Reproductive	I	6.9E-07	2.4E-03	H
95-50-1	Dichlorobenzene, 1,2-	2.0E-01	Body weight	H	NA	NA	
106-46-7	Dichlorobenzene, 1,4-	8.0E-01	Reproductive	I	NA	NA	
75-71-8	Dichlorodifluoromethane	2.0E-01	Liver	H	NA	NA	
107-06-2	Dichloroethane, 1,2-	NA			2.6E-05	9.1E-02	I
75-35-4	Dichloroethylene, 1,1-	NA			5.0E-05	1.8E-01	I
78-87-5	Dichloropropane, 1,2-	4.0E-03	Respiratory	I	NA	NA	
10061-01-5	Dichloropropene, cis-1,3-	2.0E-02	Respiratory	I	3.7E-05	1.3E-01	H
10061-02-6	Dichloropropene, trans-1,3-	2.0E-02	Respiratory	I	3.7E-05	1.3E-01	H
57-97-6	Dimethylbenz(a)anthracene, 7,12-	NA			2.4E-02	8.4E+01	D
68-12-2	Dimethylformamide, N,N-	3.0E-02	Liver	I	NA	NA	
95-65-8	Dimethylphenol, 3,4-	NA	NA		NA	NA	
121-14-2	Dinitrotoluene, 2,4-	NA			1.9E-04	6.8E-01	D
123-91-1	Dioxane, 1,4-	8.0E-01	No liver, kidney, or hemato effects	D	NA	NA	
122-66-7	Diphenylhydrazine, 1,2-	NA			2.2E-04	7.7E-01	I
106-89-8	Epichlorohydrin	1.0E-03	Respiratory	I	1.2E-06	4.2E-03	I
106-88-7	Epoxybutane, 1,2-	2.0E-02	Respiratory	I	NA	NA	
111-15-9	Ethoxyethanol acetate, 2-	3.0E-01	Reproductive (male), hemato	D	NA	NA	
110-80-5	Ethoxyethanol, 2-	2.0E-01	Reproductive	I	NA	NA	
100-41-4	Ethylbenzene	1.0E+00	Developmental	I	NA	NA	
106-93-4	Ethylene dibromide	2.0E-04	Reproductive	H	2.2E-04	7.7E-01	I
107-21-1	Ethylene glycol	6.0E-01	Respiratory	D	NA	NA	
75-21-8	Ethylene oxide	NA			1.0E-04	3.5E-01	H
50-00-0	Formaldehyde	NA			1.3E-05	4.6E-02	I
98-01-1	Furfural	5.0E-02	Respiratory	H	NA	NA	
87-68-3	Hexachloro-1,3-butadiene	NA			2.2E-05	7.7E-02	I
118-74-1	Hexachlorobenzene	NA			4.6E-04	1.6E+00	I
77-47-4	Hexachlorocyclopentadiene	7.0E-05	Respiratory	H	NA	NA	
67-72-1	Hexachloroethane	NA			4.0E-06	1.4E-02	I
110-54-3	Hexane, -	2.0E-01	Respiratory and neurological	I	NA	NA	

(continued)

Table 6-1. (continued)

CAS#	Name	Noncarcinogens			Carcinogens		
		RfC (mg/m ³)	RfC Target Organ	Ref ^a	Inhal URF (µg/m ³) ¹	Inhal CSF (mg/kg/d) ¹	Ref ^a
78-59-1	Isophorone	4.0E-03	Body weight	FR	NA	NA	
7439-96-5	Manganese	5.0E-05	Neurological	I	NA	NA	
7439-97-6	Mercury	3.0E-04	Neurological	I	NA	NA	
67-56-1	Methanol	1.3E+01	Developmental	D	NA	NA	
110-49-6	Methoxyethanol acetate, 2-	3.0E-02	Reproductive (male)	D	NA	NA	
109-86-4	Methoxyethanol, 2-	2.0E-02	Reproductive	I	NA	NA	
74-83-9	Methyl bromide (bromomethane)	5.0E-03	Respiratory	I	NA	NA	
74-87-3	Methyl chloride (chloromethane)	NA			1.8E-06	6.3E-03	H
78-93-3	Methyl ethyl ketone	1.0E+00	Developmental	I	NA	NA	
108-10-1	Methyl isobutyl ketone	8.0E-02	Kidney and liver	H	NA	NA	
80-62-6	Methyl methacrylate	7.0E-01	Respiratory	I	NA	NA	
1634-04-4	Methyl <i>tert</i> -butyl ether	3.0E+00	Kidney and liver	I	NA	NA	
56-49-5	Methylcholanthrene, 3-	NA			2.1E-03	7.4E+00	D
75-09-2	Methylene chloride	3.0E+00	Liver	H	4.7E-07	1.6E-03	I
91-20-3	Naphthalene	1.0E-02	Respiratory	A	NA	NA	
7440-02-0	Nickel	NA			2.4E-04	8.4E-01	I
98-95-3	Nitrobenzene	2.0E-03	Kidney, liver, hematological, adrenal	H	NA	NA	
79-46-9	Nitropropane, 2-	2.0E-02	Liver	I	2.7E-03	9.4E+00	H
55-18-5	Nitrosodiethylamine	NA			4.30E-02	1.5E+02	I
924-16-3	Nitrosodi- <i>n</i> -butylamine	NA			1.60E-03	5.6E+00	I
930-55-2	<i>n</i> -Nitrosopyrrolidine	NA			6.10E-04	2.1E+00	I
108-95-2	Phenol	2.0E-02	No effects	FR	NA	NA	
85-44-9	Phthalic anhydride	1.2E-01	Respiratory	H	NA	NA	
75-56-9	Propylene oxide	3.0E-02	Respiratory	I	3.7E-06	1.3E-02	I
110-86-1	Pyridine	7.0E-03	Liver	O	NA	NA	
100-42-5	Styrene	1.0E+00	Neurological	I	NA	NA	
1746-01-6	TCDD, 2,3,7,8-	NA			NA	1.6E+05	H
630-20-6	Tetrachloroethane, 1,1,1,2-	NA			7.4E-06	2.6E-02	I
127-18-4	Tetrachloroethylene	3.0E-01	Neurological	A	NA	NA	
79-34-5	Tetrachloroethane, 1,1,2,2-	NA			5.8E-05	2.0E-01	I

(continued)

Table 6-1. (continued)

CAS#	Name	Noncarcinogens			Carcinogens		
		RfC (mg/m ³)	RfC Target Organ	Ref ^a	Inhal URF (μg/m ³) ⁻¹	Inhal CSF (mg/kg/d) ⁻¹	Ref ^a
108-88-3	Toluene	4.0E-01	Respiratory and neurological	I	NA	NA	
95-53-4	Toluidine, o-	NA			6.9E-05	2.4E-01	D
76-13-1	Trichloro-1,2,2-trifluoroethane, 1,1,2-	3.0E+01	Body weight	H	NA	NA	
120-82-1	Trichlorobenzene, 1,2,4-	2.0E-01	Liver	H	NA	NA	
71-55-6	Trichloroethane, 1,1,1-	1.0E+00	Neurological	SF	NA	NA	
79-00-5	Trichloroethane, 1,1,2-	NA			1.6E-05	5.6E-02	I
79-01-6	Trichloroethylene	NA			1.7E-06	6.0E-03	SF
75-69-4	Trichlorofluoromethane	7.0E-01	Kidney and respiratory	H	NA	NA	
121-44-8	Triethylamine	7.0E-03	No respiratory effects	I	NA	NA	
7440-62-2	Vanadium	7.0E-05	Respiratory	D	NA	NA	
108-05-4	Vinyl acetate	2.0E-1	Respiratory	I	NA	NA	
75-01-4	Vinyl chloride	NA			8.4E-05	3.0E-01	H
1330-20-7	Xylenes (total)	3.0E-01	Neurological	A	NA	NA	

CAS = Chemical Abstract Service.

CSF = Cancer slope factor.

NA = Not available.

RfC = Reference concentration.

URF = Unit risk factor.

^a Sources:

I = IRIS (U.S. EPA, 1998)

H = HEAST (U.S. EPA, 1997a)

A = Agency for Toxic Substances Disease Registry (ATSDR) minimal risk levels (MRLs)

SF = Superfund Risk Issue Paper (U.S. EPA, 1996c; U.S. EPA, n.d.)

FR = 61 FR 42317-354 (U.S. EPA, 1996b)

D = Developed for this study.

O = Other source (see Sections 6.1.1 and 6.1.2).

For acetone, naphthalene, tetrachloroethylene, and total xylenes, ATSDR's chronic inhalation MRLs were used. Naphthalene is currently undergoing review by EPA's IRIS pilot program (future publication date not known) and a new RfC may be available soon. Provisional RfCs were identified for cyclohexanol, isophorone, and phenol in a *Federal Register* notice (61 FR 42317) concerning solvents listings (U.S. EPA, 1996b). An inhalation ADI was identified for pyridine (U.S. EPA, 1986). An RfC for 1,1,1-trichloroethane was identified in a Superfund risk issue paper (U.S. EPA, 1996c). Table 6-2 summarizes the alternate RfCs identified for this analysis, as well as the target organs, sources, and critical studies.

Table 6-2. Alternate Chronic Inhalation Health Benchmarks

CAS #	Chemical Name	Inhalation Benchmark and Benchmark Value	Target Organ	Source
67-64-1	Acetone (2-propanone)	RfC = 13 ppm (31 mg/m ³)	Neurological	ATSDR chronic inhal MRL based on Stewart et al. 1975. <i>Acetone: Development of a Biological Standard for the Industrial Worker by Breath Analysis</i> , Cincinnati, OH: NIOSH. NTIS PB82-172917
108-93-0	Cyclohexanol	Provisional RfC = 0.00006 mg/m ³	Muscle	61 FR 42343 (U.S. EPA, 1996b); standard RfC methodology
78-59-1	Isophorone	Provisional RfC= 0.004 mg/m ³	Body weight	61 FR 42345 (U.S. EPA, 1996b); standard RfC methodology
91-20-3	Naphthalene	RfC = 0.002 ppm (0.01 mg/m ³)	Respiratory	ATSDR chronic inhal MRL based on NTP TR-410 (1992); value may change because naphthalene is undergoing review in the IRIS pilot program
108-95-2	Phenol	Provisional RfC = 0.02 mg/m ³	No effects	61 FR 42336 (U.S. EPA, 1996b); standard RfC methodology
110-86-1	Pyridine	Inhalation ADI= 0.002 mg/kg/d; converts to 0.007 mg/m ³	Liver	Cited in Health and Environmental Effects Profile (HEEP) for Pyridine (EPA/600/x-86-168)
127-18-4	Tetrachloroethylene	RfC = 0.04 ppm (0.3 mg/m ³)	Neurological	ATSDR chronic inhal MRL based on Ferroni et al. 1992. Neurobehavioral and neuroendocrine effects of occupational exposure to perchloroethylene. <i>Neurotoxicology</i> 12: 243-247
71-55-6	1,1,1-Trichloroethane	RfC= 1.0 mg/m ³	Neurological	Superfund risk issue paper (U.S. EPA 1996c)
1330-20-7	Xylenes (total)	RfC = 0.1 ppm (0.3 mg/m ³)	Neurological	ATSDR chronic inhal MRL based on Uchida et al., 1993. Symptoms and signs in workers exposed predominantly to xylenes. <i>Int Arch Occup Environ Health</i> 64:597-605.

6.1.2 Chronic Inhalation Health Benchmarks Derived for This Study

Chronic inhalation health benchmarks were developed for constituents lacking IRIS, HEAST, alternative EPA, or ATSDR values. RfCs were developed for

- 2-Chlorophenol
- Cobalt
- Cresols

- 1,4-Dioxane
- 2-Ethoxyethanol acetate
- Ethylene glycol
- Methanol
- 2-Methoxyethanol acetate
- Vanadium.

For cobalt, cresols, 1,4-dioxane, ethylene glycol, and methanol, appropriate inhalation studies were identified and RfCs were developed using EPA's standard RfC methodology as detailed in *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994d). For vanadium, the study that the ATSDR acute inhalation MRL is based on was used but was adjusted for chronic exposure. For 2-chlorophenol, an RfC was developed using route-to-route extrapolation of the oral RfD for 2-chlorophenol (U.S. EPA, 1998). RfCs were derived for 2-ethoxyethanol acetate and 2-methoxyethanol acetate based on RfCs for 2-ethoxyethanol and 2-methoxyethanol, respectively.

Inhalation unit risk factors and inhalation cancer slope factors were developed for

- Bromodichloromethane
- Chlorodibromomethane
- 7,12-Dimethylbenz[*a*]anthracene
- 2,4-Dinitrotoluene
- 3-Methylcholanthrene
- o-Toluidine.

For bromodichloromethane, chlorodibromomethane, 2,4-dinitrotoluene, and o-toluidine, the oral CSFs (U.S. EPA, 1997a, 1998) were used to develop inhalation URFs for the compounds. For 7,12-dimethylbenz[*a*]anthracene and 3-methylcholanthrene, inhalation URFs developed by California's EPA (1997b) were used as the cancer benchmarks.

EPA examined the toxicity data for 3,4-dimethylphenol and determined that it was not appropriate to derive an RfC. Very little toxicity or metabolism data specific to 3,4-dimethylphenol are available. Although an RfD has been developed by EPA (U.S. EPA, 1998), route-to-route extrapolation is not recommended because of the potential for respiratory tract effects following inhalation exposure and "first-pass" effects following ingestion exposure.

Table 6-3 summarizes the RfCs, inhalation unit risk factors, and inhalation cancer slope factors derived for use in the air characteristic analysis, the method of development and critical studies used, and the target organs identified. Details on the derivation of these inhalation benchmark values are provided in Appendix E.

Table 6-3. Chronic Inhalation Health Benchmarks Derived for This Study

CAS #	Chemical Name	Inhalation Benchmark and Benchmark Value	RfC Target Organ	Method of Derivation
75-27-4	Bromodichloromethane (dichlorobromomethane)	Inhal CSF = 6.2E-02 per mg/kg/d Inhal URF = 1.8E-05 per $\mu\text{g}/\text{m}^3$		Inhal CSF and URF based on IRIS oral CSF (renal)
124-48-1	Chlorodibromomethane (dibromochloromethane)	Inhal CSF = 8.4E-02 per mg/kg/d Inhal URF = 2.4E-05 per $\mu\text{g}/\text{m}^3$		Inhal CSF and URF based on IRIS oral CSF (hepatocellular adenoma/carcinoma)
95-57-8	2-Chlorophenol (o-)	RfC = 0.0014 mg/m^3	Repro/developmental	Route-to-route extrapolation of IRIS RfD (0.005 $\text{mg}/\text{kg}/\text{d}$ for reproductive effects)
7440-48-4	Cobalt	RfC = 0.00001 mg/m^3	Respiratory	Standard RfC derivation based on NTP TR-471 (1996a); supported by: ATSDR intermediate inhal MRL = 3E-05 mg/m^3 based on NTP TR-TOX-5 (1991) and CalEPA RfC = 5E-06 mg/m^3 based on Bucher et al. 1990. Inhalation toxicity studies of cobalt sulfate in F344/N rats and B6C3F1 mice. <i>Fundam Appl Toxicol</i> 15:357-372.
1319-77-3	Cresols, total	RfC = 0.0004 mg/m^3	Hematological	Standard RfC derivation based on: Uzhdavini ER, Astaf'yeva K, Mamayeva AA, Bakhtizina GZ. 1972. [Inhalation toxicity of o-cresol]. Trudy Ufimskogo Nauchno-Issledovatel'skogo Instituto Gigiyeny Profzabolevaniya, 7:115-9. (Russian)
57-97-6	7,12-Dimethylbenz[a]anthracene	Inhal CSF = 8.4E+01 per mg/kg/d Inhal URF = 2.4E-02 per $\mu\text{g}/\text{m}^3$		Inhal CSF and URF derived by CalEPA (1997) based on TD_{50} approach
95-65-8	3,4-Dimethylphenol	NA - RfC derivation is inappropriate		
121-14-2	2,4-Dinitrotoluene	Inhal CSF = 6.8E-01 per mg/kg/d Inhal URF = 1.9E-04 per $\mu\text{g}/\text{m}^3$		Inhal CSF and URF based on IRIS oral CSF (liver, mammary gland)
123-91-1	1,4-Dioxane (1,4-diethyleneoxide)	RfC = 0.8 mg/m^3	Liver, kidney, hematological	Standard RfC derivation based on Torkelson et al. 1974. 1,4-Dioxane. II. Results of a 2-year inhalation study in rats. <i>Toxicol Appl Pharmacol</i> 30:287-298.

(continued)

Table 6-3. (continued)

CAS #	Chemical Name	Inhalation Benchmark and Benchmark Value	RfC Target Organ	Method of Derivation
111-15-9	2-Ethoxyethanol acetate	RfC= 0.3 mg/m ³	Repro (male)/ hematological	Derived from RfC for ethoxyethanol
107-21-1	Ethylene glycol	RfC= 0.6 mg/m ³	Respiratory	Derived using standard RfC methodology
67-56-1	Methanol	RfC = 13 mg/m ³	Developmental	Standard RfC derivation based on Rogers et al. 1993. The developmental toxicity of inhaled methanol in the CD-1 mouse, with quantitative dose-response modeling for estimation of benchmark doses. <i>Teratology</i> 47(3):175-188.
110-49-6	2-Methoxyethanol acetate	RfC= 0.03 mg/m ³	Reproductive (male)	Derived from RfC for methoxyethanol
56-49-5	3-Methylcholanthrene	Inhal CSF = 7.4E+00 per mg/kg/d Inhal URF = 2.1E-03 per µg/m ³		Inhal CSF and URF derived by CalEPA (1997) based on TD ₅₀ approach
95-53-4	o-Toluidine	Inhal CSF = 2.4E-01 per mg/kg/d Inhal URF = 6.9E-05 per µg/m ³		Inhal CSF and URF based on HEAST oral CSF (skin fibroma)
7440-62-2	Vanadium	RfC = 0.00007 mg/m ³	Respiratory	RfC based on ATSDR acute inhal MRL study, but adjusted for chronic exposure. Zenz and Berg 1967. Human responses to controlled vanadium pentoxide exposure. <i>Arch Environ Health</i> 14:709-12. (acute inhal MRL = 0.0002 mg/m ³)

6.2 Subchronic Inhalation Health Benchmarks

Information on intermediate or subchronic inhalation noncancer benchmark values for constituents considered in this study is summarized in Table 6-4. Data collected included subchronic RfCs and ATSDR intermediate inhalation MRLs. Figure 6-2 describes the approach used to select the subchronic inhalation health benchmarks used in this analysis.

ATSDR intermediate inhalation MRLs are for use with exposure durations of 15 to 364 days and are derived from subchronic toxicological or epidemiological studies. Subchronic inhalation RfCs were identified in HEAST (U.S. EPA, 1997a) or were derived from existing RfCs or chronic inhalation MRLs. A number of chronic RfCs cited in IRIS or HEAST were based on subchronic studies; for these constituents, the uncertainty factor (UF) applied to extrapolate from subchronic to chronic duration (usually 10) for the derivation of the chronic RfC was removed, resulting in subchronic RfCs. Five RfCs cited in IRIS were based on chronic studies; for these constituents, a modifying factor (MF) of 10 was applied to extrapolate from chronic to subchronic duration to derive subchronic RfCs. Chronic MRLs were available for 1,2-dichloroethane and nickel (0.2 ppm and $2.0\text{E-}4\text{ mg/m}^3$, respectively); a modifying factor was used to extrapolate from chronic to subchronic duration, resulting in interim subchronic RfCs (8.1 mg/m^3 and $2.0\text{E-}3\text{ mg/m}^3$, respectively). RfCs derived for 1,4-dioxane, naphthalene, and tetrachloroethylene were based on chronic inhalation studies; a modifying factor was applied to these RfCs to extrapolate from chronic to subchronic duration. Seventy-five subchronic RfCs or intermediate inhalation MRLs were identified for the 105 selected constituents; however,

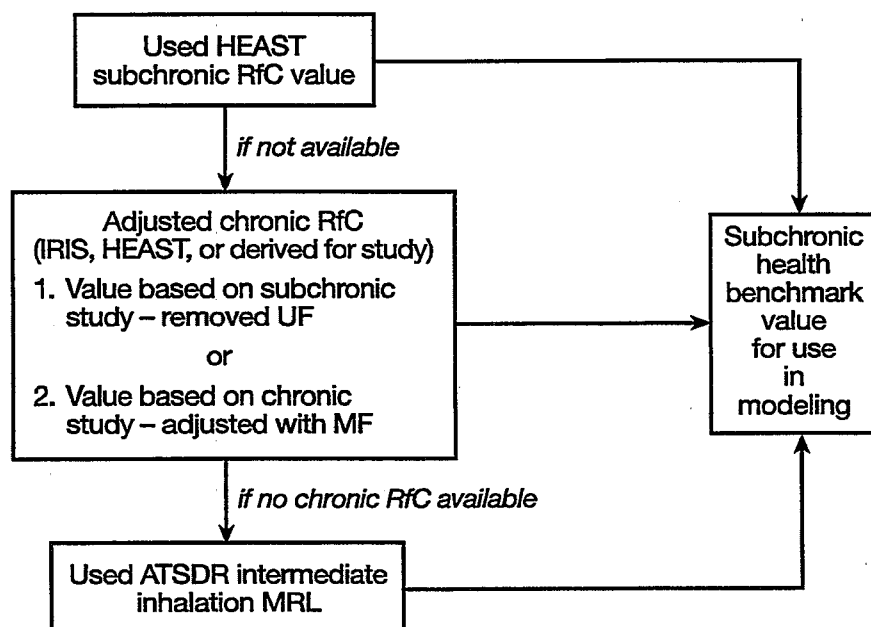


Figure 6-2. Approach used to select subchronic noncancer inhalation benchmark values.

Table 6-4. Subchronic Inhalation Health Benchmarks

CAS #	Name	Subchronic RfC (mg/m ³)	Target Organ	Source
75-07-0	Acetaldehyde	9.0E-02	Respiratory	RfC based on subchronic study - removed UF
67-64-1	Acetone	3.1E+01	Neurological	ATSDR intermediate MRL
75-05-8	Acetonitrile	5.0E-01	Liver	HEAST
107-02-8	Acrolein	2.0E-04	Respiratory	RfC based on subchronic study - removed UF
79-10-7	Acrylic acid	3.0E-03	Respiratory	HEAST
107-13-1	Acrylonitrile	2.0E-02	Respiratory	RfC based on chronic study - adjusted w/ MF
107-05-1	Allyl chloride	1.0E-02	Neurological	HEAST
62-53-3	Aniline	1.0E-02	Spleen	HEAST
7440-39-3	Barium	5.0E-03	Developmental	HEAST
71-43-2	Benzene	1.3E-02	Neurological	ATSDR intermediate MRL
75-15-0	Carbon disulfide	7.0E-01	Neurological	HEAST
56-23-5	Carbon tetrachloride	3.1E-01	Liver	ATSDR intermediate MRL
126-99-8	Chloro-1,3-butadiene, 2-(chloroprene)	7.0E-02	Respiratory	HEAST
108-90-7	Chlorobenzene	2.0E-01	Liver, kidney	Superfund Risk Assessment Issue Paper
67-66-3	Chloroform	2.4E-01	Liver	ATSDR intermediate MRL
7440-47-3	Chromium VI	2.0E-05	Respiratory	ATSDR intermediate MRL
7440-48-4	Cobalt	3.0E-05	Respiratory	ATSDR intermediate MRL
1319-77-3	Cresols (total)	1.0E-04	Hematological	RfC based on subchronic study - removed UF
98-82-8	Cumene	4.0E+00	Kidney, adrenal	RfC based on subchronic study - removed UF
108-93-0	Cyclohexanol	6.0E-04	Muscle	RfC based on subchronic study - removed UF
96-12-8	Dibromo-3-chloropropane, 1,2-	2.0E-03	Reproductive	RfC based on subchronic study - removed UF
95-50-1	Dichlorobenzene, 1,2-	2.0E+00	Body wt	HEAST
106-46-7	Dichlorobenzene, 1,4-	2.5E+00	Liver	HEAST
75-71-8	Dichlorodifluoromethane	2.0E+00	Liver	HEAST
107-06-2	Dichloroethane, 1,2-	8.1E+00	Liver	chronic MRL avail. - adjusted w/ MF
75-35-4	Dichloroethylene, 1,1-	7.9E-02	Liver	ATSDR intermediate MRL
78-87-5	Dichloropropane, 1,2-	1.3E-02	Respiratory	HEAST
10061-01-5	Dichloropropene, cis-1,3-	2.0E-02	Respiratory	HEAST
10061-02-6	Dichloropropene, trans-1,3-	2.0E-02	Respiratory	HEAST

(continued)

Table 6-4. (continued)

CAS#	Name	Subchronic RfC (mg/m ³)	Target Organ	Source
68-12-2	Dimethylformamide, <i>N,N</i> -	3.0E-02	Liver, GI	HEAST
123-91-1	Dioxane, 1,4-	8.0E+00	No liver, kidney, or hemato effects	RTI-derived RfC based on chronic study - adjusted w/ MF
106-89-8	Epichlorohydrin	1.0E-02	Respiratory	HEAST
106-88-7	Epoxybutane, 1,2-	2.0E-01	Respiratory	RfC based on chronic study - adjusted w/ MF
110-80-5	Ethoxyethanol, 2-	2.0E+00	Hematological	HEAST
100-41-4	Ethylbenzene	8.7E-01	Developmental	ATSDR intermediate MRL
106-93-4	Ethylene dibromide	2.0E-03	Reproductive	HEAST
107-21-1	Ethylene glycol	6.0E+00	Respiratory	RfC based on subchronic study - removed UF
75-21-8	Ethylene oxide	1.6E-01	Kidney	ATSDR intermediate MRL
50-00-0	Formaldehyde	1.2E-02	Respiratory	ATSDR intermediate MRL
98-01-1	Furfural	5.0E-01	Respiratory	HEAST
77-47-4	Hexachlorocyclopentadiene	7.0E-04	Respiratory	HEAST
67-72-1	Hexachloroethane	5.8E+01	Neurological	ATSDR intermediate MRL
110-54-3	Hexane, -	2.0E-01	Neurological	HEAST
78-59-1	Isophorone	4.0E-02	Body weight	RfC based on subchronic study - removed UF
7439-96-5	Manganese	5.0E-04	Neurological	RfC based on chronic study - adjusted w/ MF
7439-97-6	Mercury	3.0E-04	Neurological	HEAST
109-86-4	Methoxyethanol, 2-	2.0E-01	Reproductive	HEAST
74-83-9	Methyl bromide (bromomethane)	1.9E-01	Neurological	ATSDR intermediate MRL
74-87-3	Methyl chloride (chloromethane)	4.1E-01	Liver	ATSDR intermediate MRL
78-93-3	Methyl ethyl ketone	1.0E+00	Developmental	HEAST
108-10-1	Methyl isobutyl ketone	8.0E-01	Liver, kidney	HEAST
80-62-6	Methyl methacrylate	7.0E+00	Respiratory	RfC based on chronic study - adjusted w/ MF
1634-04-4	Methyl <i>tert</i> -butyl ether	2.1E+00	Neurological	ATSDR intermediate MRL
75-09-2	Methylene chloride	3.0E+00	Liver	HEAST
91-20-3	Naphthalene	1.0E-01	Respiratory	RTI-derived RfC based on chronic study - adjusted w/ MF

(continued)

CAS #	Name	Subchronic RfC (mg/m ³)	Target Organ	Source
7440-02-0	Nickel	2.0E-03	Respiratory	chronic MRL avail. - adjusted w/ MF
98-95-3	Nitrobenzene	2.0E-02	Hemato, adrenal, kidney, liver	HEAST
79-46-9	Nitropropane, 2-	2.0E-02	Liver	HEAST
108-95-2	Phenol	2.0E-01	No effects	RfC based on subchronic study - removed UF
85-44-9	Phthalic anhydride	1.2E-01	Respiratory	HEAST
75-56-9	Propylene oxide	3.0E-02	Respiratory	HEAST
100-42-5	Styrene	3.0E+00	Neurological	HEAST
79-34-5	Tetrachloroethane, 1,1,2,2-	2.7E+00	Liver	ATSDR intermediate MRL
127-18-4	Tetrachloroethylene	3.0E+00	Neurological	RTI-derived RfC based on chronic study - adjusted w/ MF
108-88-3	Toluene	4.0E+00	Respiratory, neurological	RfC based on chronic study - adjusted w/ MF
76-13-1	Trichloro-1,2,2- trifluoroethane, 1,1,2-	3.0E+01	Body wt	HEAST
120-82-1	Trichlorobenzene, 1,2,4-	2.0E+00	Liver	HEAST
71-55-6	Trichloroethane, 1,1,1-	3.8E+00	Neurological	ATSDR intermediate MRL
79-01-6	Trichloroethylene	5.4E-01	Neurological	ATSDR intermediate MRL
75-69-4	Trichlorofluoromethane	7.0E+00	Kidney, respiratory	HEAST
121-44-8	Triethylamine	7.0E-02	No effects	RfC based on subchronic study - removed UF
7440-62-2	Vanadium	7.0E-04	Respiratory	RfC based on subchronic study - removed UF
108-05-4	Vinyl acetate	2.0E-01	Respiratory	HEAST
75-01-4	Vinyl chloride	7.7E-02	Liver	ATSDR intermediate MRL
1330-20-7	Xylenes (total)	3.0E+00	Developmental	ATSDR intermediate MRL

because subchronic exposures were modeled only for LAUs and wastepiles for volatile chemicals and metals, only 64 of these benchmarks were used. The remaining 11 were for semivolatiles not needed in LAUs and wastepiles..

6.3 Acute Inhalation Health Benchmarks

Acute noncancer inhalation benchmark values for constituents considered in this study are summarized in Tables 6-5 and 6-6. Acute benchmarks identified include ATSDR acute inhalation MRLs, EPA acute exposure guideline levels (AEGLs), and CalEPA (1995) 1-h acute

inhalation reference exposure levels (RELs). Twenty-three acute ATSDR MRLs were identified. ATSDR acute inhalation MRLs are for use with exposure durations of 14 days or less and are derived from acute toxicological or epidemiological studies (see Table 6-5). AEGLs have been derived for aniline and ethylene oxide. CalEPA (1995) derived 1-h acute inhalation RELs for 29 of the 105 constituents. ATSDR acute MRLs were considered most appropriate for use in modeling, therefore CalEPA acute inhalation RELs were used only when MRLs were unavailable (for 15 constituents, see Table 6-6). Figure 6-3 describes the approach used to select the acute inhalation health benchmarks used in this analysis.

CalEPA's RELs can be applied in risk characterization for routine industrial emissions and planned releases as well as unplanned releases. These acute RELs are similar to EPA's AEGLs; both incorporate the recommendations of the National Academy of Science (NAS) published in *Guidelines for Developing Community Emergency Exposure Levels for Hazardous*

Table 6-5. ATSDR Acute Inhalation MRLs

CAS #	Name	ATSDR acute inhal MRL (mg/m ³)	Target Organ
67-64-1	Acetone	6.2E+01	Neurological
107-02-8	Acrolein	1.1E-04	Ocular
107-13-1	Acrylonitrile	2.2E-01	Neurological
71-43-2	Benzene	1.6E-01	Immunological
56-23-5	Carbon tetrachloride	1.3E+00	Liver
67-66-3	Chloroform	4.9E-01	Liver
106-46-7	Dichlorobenzene, 1,4-	4.8E+00	Developmental
107-06-2	Dichloroethane, 1,2-	8.1E-01	Immunological
78-87-5	Dichloropropane, 1,2-	2.3E-01	Respiratory
107-21-1	Ethylene glycol	1.3E+00	Kidney
50-00-0	Formaldehyde	6.1E-02	Respiratory
67-72-1	Hexachloroethane	5.8E+01	Neurological
74-83-9	Methyl bromide (bromomethane)	1.9E-01	Neurological
74-87-3	Methyl chloride (chloromethane)	1.0E+00	Neurological
1634-04-4	Methyl <i>tert</i> -butyl ether	6.1E+00	Neurological
75-09-2	Methylene chloride	1.4E+00	Neurological
127-18-4	Tetrachloroethylene	1.4E+00	Neurological
108-88-3	Toluene	1.1E+01	Neurological
71-55-6	Trichloroethane, 1,1,1-	1.1E+01	Neurological
79-01-6	Trichloroethylene	1.1E+01	Neurological
7440-62-2	Vanadium	2.0E-04	Respiratory
75-01-4	Vinyl chloride	1.3E+00	Developmental
1330-20-7	Xylenes (total)	4.3E+00	Neurological

Table 6-6. CalEPA's 1-Hour Acute Inhalation Reference Exposure Levels (RELs)

CAS #	Name	CalEPA 1-h REL (mg/m ³)	Effect Level	Effect
79-10-7	Acrylic acid	6.0E+00	I	Respiratory irritation
7440-38-2	Arsenic	3.9E-04	II	Repro/developmental
75-15-0	Carbon disulfide	3.1E+00	II	Repro/developmental
123-91-1	Dioxane, 1,4-	1.8E+00	I	Eye irritation
106-89-8	Epichlorohydrin	8.0E-01	I	Eye & respir irritation
111-15-9	Ethoxyethanol acetate, 2-	3.3E-01	II	Repro/developmental
110-80-5	Ethoxyethanol, 2-	8.8E-02	II	Hemato, Repro/developmental
7439-97-6	Mercury	1.8E-03	II	Repro/developmental
67-56-1	Methanol	2.8E+00	I	Mild neuro
109-86-4	Methoxyethanol, 2-	2.3E-02	I	Hemato
78-93-3	Methyl ethyl ketone	1.5E-01	I	Respiratory irritation
7440-02-0	Nickel	1.6E-03	I	Immuno
108-95-2	Phenol	1.5E-01	I	Respiratory irritation
75-56-9	Propylene oxide	9.3E-01	I	Eye & respir irritation
100-42-5	Styrene	2.2E+01	I	Eye & respir irritation

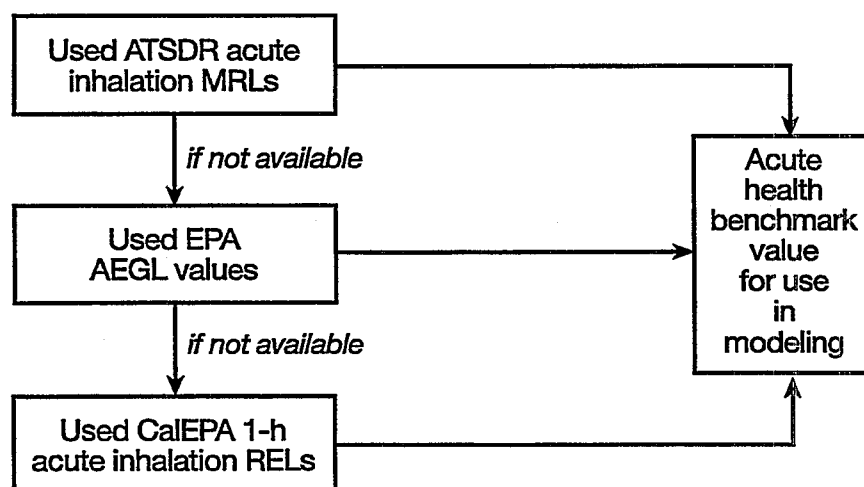


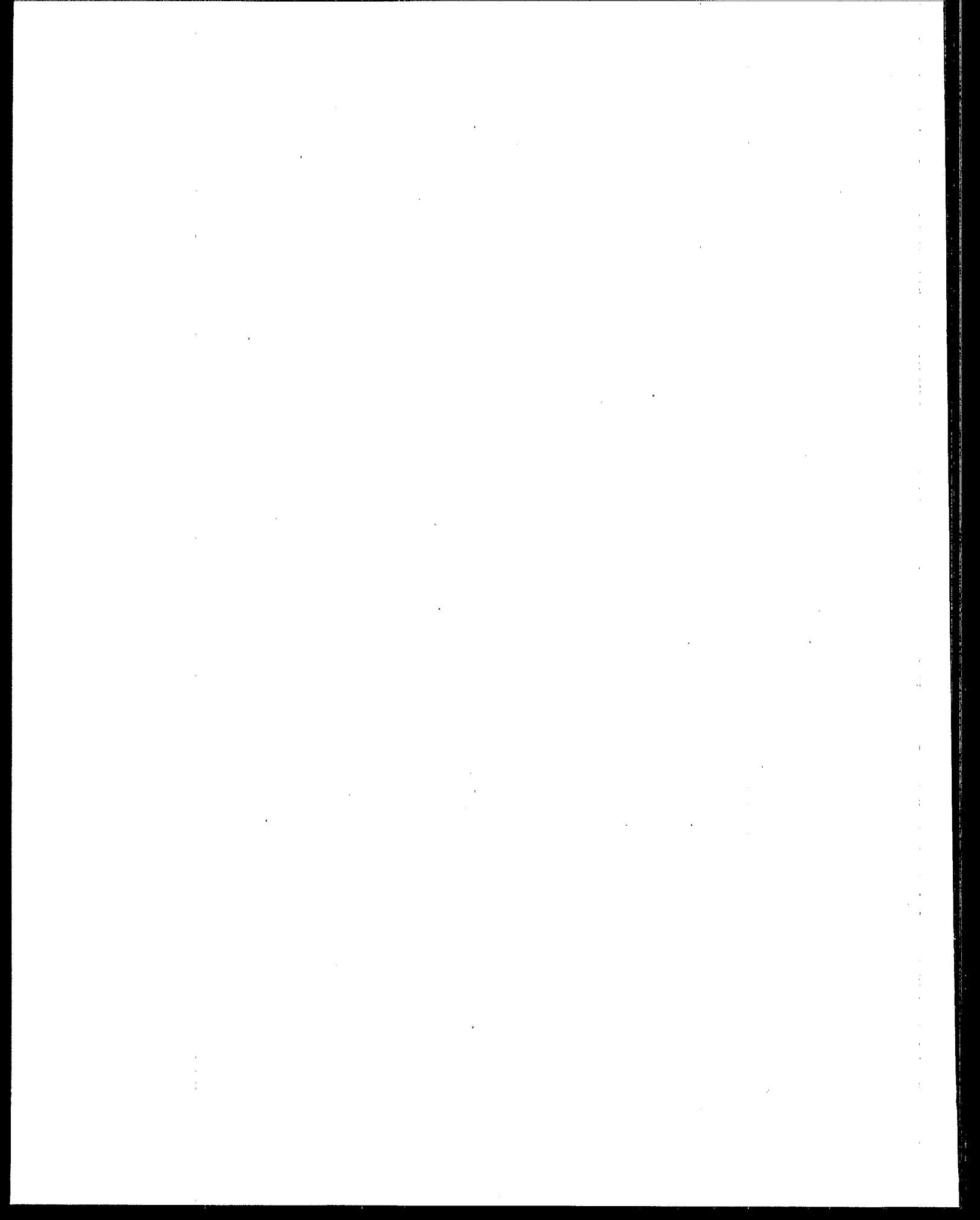
Figure 6-3. Approach used to select acute noncancer inhalation health benchmark values.

Substances (NAS, 1993). AEGLs represent short-term threshold or ceiling exposure values intended for the protection of the general public, including susceptible or sensitive individuals. The AEGLs represent biological reference values for each of four different exposure periods of 30 minutes, 1 hour, 4 hours, and 8 hours. CalEPA's acute RELs and EPA's AEGLs have been established for three grades of effect:

- Level I and AEGL-1: discomfort or mild effect level
- Level II and AEGL-2: disability or serious effect level
- Level III and AEGL-3: life-threatening effect level.

Only AEGL-1s were used in this study. The 8-h AEGL-1 for aniline is 3.8 mg/m³; no AEGL-1 levels have been derived for ethylene oxide (U.S. EPA, 1997b).

A total of 39 acute benchmarks were identified for the 105 constituents in this study; however, because acute exposures were modeled only for LAUs and wastepiles for volatile chemicals and metals, only 35 of these benchmarks were used. The remaining 4 were for semivolatiles not modeled in LAUs and wastepiles.



7.0 Development of Risk-Specific Waste Concentration Distribution

This section describes the Monte Carlo model that combines results of the emissions modeling (Section 4.0) and dispersion modeling (Section 5.0) with exposure factor distributions to calculate air concentration, risk or hazard quotient, and risk-specific waste concentration (C_w) for chronic exposures, and the model used to calculate risk-specific waste concentrations for subchronic and acute exposures. In addition, it describes modifications made to the methodology for lead.

7.1 Overview

A Monte Carlo analysis was performed in which the location of the receptor and various exposure factors (body weight, inhalation rate, and exposure duration) were varied (exposure frequency was kept fixed as it has little variability). For each constituent and waste management unit type combination, a separate Monte Carlo simulation was run for each WMU in the Industrial D Survey database (Shroeder et al., 1987). The emission rate for the specific constituent from the specific WMU was used as an input to the Monte Carlo simulation and was not varied across iterations within a simulation.

A thousand iterations were performed for each WMU, resulting in a distribution of waste concentrations (C_w) that would result in the specified risk criterion. This distribution captures the range in waste concentration attributable to the variability in the potential location and to the exposure factors associated with each receptor. From this distribution, the 85th, 90th, and 95th percentiles were selected to characterize the distribution. These percentiles represent the percentage of receptors that are protected at the risk criterion for a specific WMU.

When the Monte Carlo simulation had been run for all the WMUs, a cumulative distribution across all facilities for each protection level (85, 90, or 95 percent of receptors) was obtained for each receptor at each distance. This distribution reflects the variability across facilities. In developing this distribution, the results were weighted using the facility weights from the Industrial D Survey data. These weights indicate the number of facilities in the United States represented by a particular facility in the Industrial D Survey database. The resulting cumulative distribution accounts for variability across all facilities represented, not just those actually modeled.

Each iteration of the Monte Carlo simulation required the following steps:

- Select a receptor location

- Obtain the appropriate unitized air concentration
- Calculate air concentration
- Select exposure factors from distributions
- Obtain health benchmarks
- Calculate risk or hazard quotient
- Backcalculate risk-specific waste concentration (C_w).

These steps are discussed in more detail in the following sections.

7.2 Select Receptor Location

Dispersion coefficients were modeled for each of 7 distances and 16 directions as described in Section 5.0. The 7 distances were 0, 25, 50, 75, 150, 500, and 1,000 meters. The 16 directions were equally spaced around the WMU. Calculations were performed for each distance, and direction was allowed to vary.

Four receptors were modeled: an adult resident, a child resident, an offsite worker, and an onsite worker. The onsite worker was always located at 0 meters from the WMU. The adult and child residents and the offsite worker were assessed at all of the six offsite distances (any distance except 0 meters). All receptors could be located in any of the 16 directions.

Direction was modeled as a uniform distribution, so that a receptor had a probability of 1 in 16 (or 0.0625) of being located in any one of the 16 directions. The same direction was used for all receptors, both on- and offsite, for a particular iteration.

7.3 Obtain Unitized Air Concentrations

After on- and offsite receptor locations had been selected, UACs for those two locations were calculated. For each location, the UAC was estimated by interpolating between the UACs developed for areas immediately above and below the actual area of the unit, as follows:

$$UAC = \left(\frac{A - A_i}{A_j - A_i} \right) \times (UAC_j - UAC_i) + UAC_i \quad (7-1)$$

where

UAC = unitized air concentration for specific WMU ($[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\text{-s}]$)

A = area of specific WMU (m^2)

A_i = area modeled in dispersion modeling immediate below area of specific WMU (m^2)

A_j = area modeled in dispersion modeling immediate above area of specific WMU (m^2)

UAC_i = unitized air concentration developed for area i ($[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\text{-s}]$)

UAC_j = unitized air concentration developed for area j ($[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\text{-s}]$).

A few WMUs had areas that fell either below the smallest area or above the largest area modeled in the dispersion modeling. If the area was less than the smallest area modeled, A_j and UAC_j were set to the values for the smallest area modeled, and A_i and UAC_i were set to zero. If the area was greater than the largest area modeled, the A_i , UAC_i , A_j , and UAC_j were set to correspond to the two largest areas modeled, based on the assumption that the UAC continues to increase with the same slope above the largest area modeled.

7.4 Calculate Air Concentration

Two air concentrations, one for the onsite worker and one for the offsite receptors, were calculated from the WMU emission rate (which remains constant through all iterations of the Monte Carlo simulation for a particular WMU) and the on- and offsite dispersion coefficients. Recall that the emission rates are emissions associated with a unit waste concentration of 1 mg/kg (or 1 mg/L for tanks), so the resulting air concentration is also associated with a unit waste concentration. Air concentration was calculated as follows:

$$C_{air} = (E_{vapor} \times 10^6 \mu\text{g/g} + C_{soil} \times E_{partic} / 3600 \text{ s/h}) \times UAC \quad (7-2)$$

where

C_{air} = air concentration associated with a unit waste concentration ($[\mu\text{g}/\text{m}^3]/[\text{mg}/\text{kg}]$ or $[\mu\text{g}/\text{m}^3]/[\text{mg}/\text{L}]$)

E_{vapor} = emission rate of constituent in vapor phase ($[\text{g}/\text{m}^2\text{-s}]/[\text{mg}/\text{kg}]$ or $[\text{g}/\text{m}^2\text{-s}]/[\text{mg}/\text{L}]$)

C_{soil} = average annual soil concentration in unit ($[\text{mg}/\text{kg}]/[\text{mg}/\text{kg}] = [\mu\text{g}/\text{g}]/[\text{mg}/\text{kg}]$ or $[\mu\text{g}/\text{g}]/[\text{mg}/\text{L}]$)

E_{partic} = emission rate of particulates ($\text{g}/\text{m}^2\text{-h}$)

UAC = unitized air concentrations ($[\mu\text{g}/\text{m}^3]/[\mu\text{g}/\text{m}^2\text{-s}]$).

Note that C_{soil} and E_{partic} are both zero for tanks. Vapor emissions (E_{vapor}) are zero for nonvolatile constituents (all metals except mercury).

7.5 Select Exposure Factors

Exposure factor distributions are described in the following subsections. Values for each exposure factor (inhalation rate, body weight, and exposure duration) were selected

independently for adults, children, and workers. The same values were used for a particular iteration for both on- and offsite workers. Exposure frequency remains constant for all iterations, though it varies by receptor. All data in this section are from the draft *Exposure Factors Handbook* (U.S. EPA, 1997; hereafter, the draft EFH)

All exposure factor distributions and point estimates were developed for the following subpopulations:

- Adult residents
- Children ages 0-3 years
- Children ages 4-10 years
- Children ages 11-18 years
- Workers.

The age ranges for children were used for consistency with the data on inhalation rate in the draft EFH.

7.5.1 Inhalation Rate

To assess chronic exposures, an average daily inhalation rate is needed. Such a rate is based on inhalation values for a variety of activities being averaged together.

The inhalation rate is linearly related to exposure and risk. A twofold change in the inhalation rate results in a twofold change in the risk estimate for a carcinogen. Thus, the sensitivity of this parameter in the analysis is a function of its variability. An examination of data presented in the draft EFH on daily inhalation rates for adults, workers, and children provides a general idea of the variability in this factor. In general, the range for both adults and workers would be around a factor of 2 or 3. Children's rates are more variable.

Single values for inhalation rates are presented as recommended values for long-term dose assessments in the draft EFH. Values of 11.3 m³/d for women and 15.2 m³/d for men are representative of average inhalation rates for adults. Upper percentile values are not recommended. These values differ significantly from the 20 m³/d commonly assumed in past EPA risk assessments. Additional data are presented for other activity patterns that can be used for special subpopulations (athletes, outdoor workers), as well as to more accurately reflect the actual exposed population. No distributional data are recommended for this parameter.

Although the draft EFH provides no guidance for developing a distribution of inhalation rates, EPA is continuing to work on the development of distributions for exposure factors from the data in the draft EFH. As part of this effort, Myers et al. (1997) present two fitted distributions, one lognormal and one gamma, for inhalation rate for male and female residents in 6 age ranges (0-3 years, 4-10 years, 11-18 years, 19-30 years, 31-60 years, and >60 years). Myers et al. find that the difference between the two distributions is negligible and recommend using the lognormal distribution.

Table 7-1. Estimated Parameters for Inhalation Rate for Residents Assuming Lognormal Distribution

Age (yr)	Gender	Mean (m ³ /d)	CV (%)	Std Dev (m ³ /d)	50%ile (m ³ /d)	90%ile (m ³ /d)	95%ile (m ³ /d)	99%ile (m ³ /d)
0-3	Male	7.52	73	5.49	6.1	14.1	17.8	27.9
0-3	Female	5.75	71	4.08	4.7	10.7	13.4	20.8
4-10	Male	9.30	30	2.79	8.9	13.0	14.5	17.7
4-10	Female	8.65	31	2.68	8.3	12.1	13.5	16.6
11-18	Male	14.58	36	5.25	13.7	21.4	24.2	30.6
11-18	Female	10.76	31	3.34	10.3	15.1	16.8	20.7
19-30	Male	16.75	31	5.19	16.0	23.7	26.5	32.6
19-30	Female	11.14	30	3.34	10.7	15.6	17.3	21.2
31-60	Male	16.32	32	5.22	15.6	23.2	25.9	32.0
31-60	Female	10.95	29	3.18	10.5	15.1	16.7	20.3
>60	Male	12.69	34	4.31	12.0	18.4	20.7	25.9
>60	Female	10.44	29	3.03	10.0	14.5	16.0	19.5

Std Dev = Mean × CV.

Note: Bolded values were used in this analysis.

Source: Myers et al., 1997, Table 5.2.

The parameters of the lognormal distribution (from Table 5.2 in Myers et al.) are presented in Table 7-1. The distributions for males were used in this analysis. For adults, the values for males aged 31-60 were used; the values for males aged 19-30 are nearly identical. The specific values that were used to define the distribution for the Monte Carlo analysis are shown in bold.

The data in the draft EFH are not adequate to develop a distribution of inhalation rate for workers. Therefore, a point estimate was used for the inhalation rate for workers. Table 7-2 summarizes the values for inhalation rate for workers presented in the draft EFH. The recommended hourly average of 1.3 m³/h was used. To convert this to a daily value, an 8-h workday was assumed, yielding a daily inhalation rate for workers of 10.4 m³/d.

Table 7-2. Recommended Inhalation Rates for Workers

Activity Type	Mean (m ³ /h)	Upper Percentile (m ³ /h)
Slow activities	1.1	NA
Moderate activities	1.5	NA
Heavy activities	2.3	NA
Hourly average	1.3	3.5

NA = Not available.

Note: Bolded values were used in this analysis.

Source: U.S. EPA, 1997, Table 5-23.

7.5.2 Body Weight

Distributions for body weight were needed that were consistent with the distributions used for inhalation rate. Therefore, body weight distributions for adult males (to be used for both adult residents and workers) and male children ages 0-3, 4-10, and 11-18 years were needed.

The draft EFH has summary data on body weight for adults broken down by gender and race. The data for males of all races ages 18 to 74 years were used. These are summarized in Table 7-3. Body weight is typically assumed to be normally distributed, and the percentiles shown in Table 7-3 agree well with a normal distribution. Therefore, a normal distribution was defined using the mean and standard deviation for males 18 to 74 years of age (these values are shown in bold in the table). Unlike a lognormal distribution, which cannot contain negative values, a normal distribution can take on negative values. Given the mean and standard deviation used, this is extremely unlikely; however, the distribution was explicitly truncated at zero to ensure that negative values did not occur.

For children, the draft EFH contains mean and standard deviations of body weights for 1-year age intervals (e.g., 1 year, 2 years). These values, summarized in Table 7-4, had to be pooled into the age ranges used for inhalation rates (0-3, 4-10, and 11-18 years).

If X denotes a random variable of interest (in this case, body weight), then the variance (or standard deviation squared) of X satisfies the following:

$$\sigma^2(X) = E(X^2) - E(X) \times E(X) \quad (7-3)$$

Table 7-3. Body Weights for Males, All Races, Ages 18-74 Years (kg)

Age (years)	Sample Size	Percentiles										
		Mean	Std Dev	5 th	10 th	15 th	25 th	50 th	75 th	85 th	90 th	95 th
18-74	5,916	78.1	13.5	58.6	62.3	64.9	68.7	76.9	85.6	91.3	95.7	102.7
18-24	988	73.8	12.7	56.8	60.4	61.9	64.8	72.0	80.3	85.1	90.4	99.5
25-34	1,067	78.7	13.7	59.5	62.9	65.4	69.3	77.5	85.6	91.1	95.1	102.7
35-44	745	80.9	13.4	59.7	65.1	67.7	72.1	79.9	88.1	94.8	98.8	104.3
45-54	690	80.9	13.6	50.8	65.2	67.2	71.7	79.0	89.4	94.5	99.5	105.3
55-64	1,227	78.8	12.8	59.9	63.8	66.4	70.2	77.7	85.6	90.5	94.7	102.3
65-74	1,199	74.8	12.8	54.4	58.5	61.2	66.1	74.2	82.7	87.9	91.2	96.6

Note: Bolded values were used in this analysis.

Source: U.S. EPA, 1997, Table 7-3.

Table 7-4. Body Weights for Male Children, Ages 6 Months to 18 Years (kg)

Age (years)	Mean	Std Dev	Age (years)	Mean	Std Dev
6-11 months	9.4	1.3	10	36.4	7.7
1	11.8	1.9	11	40.3	10.1
2	13.6	1.7	12	44.2	10.1
3	15.7	2.0	13	49.9	12.3
4	17.8	2.5	14	57.1	11.0
5	19.8	3.0	15	61.0	11.0
6	23.0	4.0	16	67.1	12.4
7	25.1	3.9	17	66.7	11.5
8	28.2	6.2	18	71.1	12.7
9	31.1	6.3			

Source: U.S. EPA, 1997, Table 7-2.

where

$\sigma^2(X)$ = variance of X (the standard deviation, σ , squared)

$E(X^2)$ = mean of X^2

$E(X)$ = mean of X.

In general, if there are L finite populations to be pooled, of sizes n_1, n_2, \dots, n_L , and the mean, $E(X)$, and standard deviation, σ , are known for each population, then the mean and standard deviation of the pooled population may be found as follows. The i th population accounts for a proportion $p_i = n_i/(n_1+n_2+\dots+n_L)$ of the total. The mean of X^2 , $E(X^2)$, may be calculated for each population by solving Equation 7-3 for $E(X^2)$. Then the pooled values of $E(X)$ and $E(X^2)$ are found by averaging these quantities of the L populations, using the proportions p_i as probabilities in calculating the average:

$$E(X) = \sum_{i=1}^n p_i \times E(X_i) \quad \text{or} \quad E(X^2) = \sum_{i=1}^n p_i \times E(X_i^2) \quad (7-4)$$

Once these values are obtained, the pooled variance (and standard deviation) can be calculated from Equation 7-3.

For body weights for children, it was assumed that each population to be pooled (i.e., each 1-year age range) was of equal size N. The p_i values were therefore equal to $N/N \times L$, or $1/L$ (where L was the number of populations to be pooled). Table 7-5 summarizes the resulting mean and standard deviations of body weight for each age group. These were then used to define a normal distribution, which, like the one for adults, was truncated at zero to prevent nonsensical results.

Table 7-5. Pooled Body Weights for Children (kg)

Age Group (years)	Mean	Std Dev
0-3	12.63	2.90
4-10	25.90	7.91
11-18	57.18	15.61

7.5.3 Exposure Duration

Data on exposure duration in the draft EFH were obtained from the distributions presented for population mobility (Chapter 14.3). Because data on males were used for inhalation rate and body weight, data on males were used for exposure duration for consistency.

For adult residents, regression methods described in Myers et al. (1997) were used to fit gamma, lognormal, Weibull, and generalized gamma distributions to the tabulated percentiles from Table 14-158 in the draft EFH. The gamma model fit best of the two-parameter models and fit adequately relative to the generalized gamma model (goodness-of-fit p-values of .96, .07, and .70 for females, males, and males/females combined). Therefore, the gamma distribution was used. Table 7-6 summarizes the data from Table 14-158 of the draft EFH and the parameters of the fitted gamma distribution.

Table 14-159 of the draft EFH provides descriptive statistics on population mobility for children by age. These data are summarized here in Table 7-7. Because children were divided into three age groups, exposure duration had to be approached in a somewhat different manner for children. Instead of using a distribution for exposure duration for children in the Monte Carlo analysis, a distribution on the age of the child at the start of exposure was used, and the 90th percentile of residential occupancy period for that age was fixed as the exposure duration.

Table 7-6. Descriptive Statistics for Residential Occupancy Period for Males (years)

Statistic or Parameter	Data	Fitted Distribution
Alpha (shape)	NA	1.32
Beta (scale)	NA	8.37
Mean	11.1	11
5 th percentile	2	1
10 th percentile	2	2
25 th percentile	4	4
50 th percentile	8	8
75 th percentile	15	15
90 th percentile	24	24
95 th percentile	31	30
98 th percentile	39	39
99 th percentile	44	45

NA = Not applicable.

Source: Data column: U.S. EPA, 1997, Table 14-158.

Table 7-7. Descriptive Statistics for Population Mobility for Children (years)

Current Age (years)	Percentile					
	25	50	75	90	95	99
3	3	5	8	13	17	22
6	4	7	10	15	18	22
9	5	8	12	16	18	22
12	5	9	13	16	18	23
15	5	8	12	16	18	23
18	4	7	11	16	19	23

Source: U.S. EPA, 1997, Table 14-159.

Exposure was constrained to begin at the beginning of one of the age ranges (so at 0, 4, or 11 years). Each of the three ages had a probability assigned to it reflecting the relative number of years in that age range. Those probabilities and the 90th percentile residential occupancy period used are summarized in Table 7-8. When an exposure duration was longer than the age range, exposure was continued in the next age range until the exposure duration or age 18 was reached. For example, if exposure began at 0 years, then 3 years was modeled as age 0 to 3, 7 years was modeled as age 4 to 10, and 3 years was modeled as 11 to 18, for a total of 13 years.

For workers, the typical default exposure values used in the past were an 8-h shift, 240 d/wk, for 40 years. The draft EFH presents data on occupational mobility that are in stark contrast to the assumed value of 40 years at a single place of employment. As presented in the draft EFH, the median occupational tenure of the working population (109.1 million people) ages 16 years of age and older in January 1987 was 6.6 years. Since the occupational tenure varies significantly according to age, the draft EFH recommends using age-dependent values. When age cannot be determined, use of the median tenure value of 6.6 years for working men and women 16 years and older is recommended.

For this study, it was not possible to find age distribution data for people employed in the waste management sector. Nor was there sufficient information in the draft EFH to develop a distribution for exposure duration. Therefore, the average of 6.6 years was used.

Table 7-8. Distribution of Age at Start of Exposure and Exposure Duration for Children

Age Range (years)	Years in Range	Probability	Exposure Duration (years)
0-3	3	0.17	13
4-10	7	0.39	15
11-18	8	0.44	16

Source: U.S. EPA, 1997, Table 14-159.

7.5.4 Exposure Frequency

Exposure frequency is the number of days per year that a receptor is exposed. It is not expected to vary much, so no distributions were developed for exposure frequency. A value of 350 d/yr was used for adult and child residents, and a value of 240 d/yr was used for workers. These are based, respectively, on 7 d/wk and 5 d/wk for 50 wk/yr and account for the receptor being elsewhere on vacation for 2 wk/yr.

7.6 Obtain Health Benchmarks

Standard health benchmarks (cancer slope factors for carcinogens and reference concentrations for noncarcinogens) were obtained for each constituent (see Section 6). The draft EFH (U.S. EPA, 1997) cautions that care must be taken that the assumptions about population parameters in the exposure analysis are consistent with the population parameters used in the dose-response analysis used to develop these health benchmarks. If the exposure scenario is not consistent with the standard factors applied in developing the dose-response relationships, then adjustment must be made for the dose-response relationship to reflect the exposure being modeled.

7.6.1 Carcinogens

In the IRIS derivation of cancer dose-response (D-R) assessments, a standard exposure scenario is assumed in calculating the slope factor (i.e., human cancer risk per unit dose) on the basis of either animal bioassay data or human data. This standard scenario has traditionally been assumed to be typical of the U.S. population and includes a body weight of 70 kg, an inhalation rate of 20 m³/d, and a lifetime of 70 years. The use of these specific values has depended on whether the slope factor was derived from animal or human epidemiological data.

For D-R studies based on animal data, the animal dose is scaled to human equivalent doses using a human body weight assumption of 70 kg. No explicit lifetime adjustment is necessary because the assumption is made that events occurring in the lifetime animal bioassay will occur with equal probability in a human lifetime, whatever that might happen to be.

For D-R studies based on human studies (either occupational or general population), the Agency has usually made no explicit assumption of body weight or human lifetime. For both of these parameters, there is an implicit assumption that the population usually of interest has the same descriptive parameters as the population analyzed by the Agency. In the rare situation where this assumption is known to be wrong, the Agency has made appropriate corrections so that the D-R parameters represent the national average population.

The draft EFH provides a table of correction factors for the D-R values tabulated in the IRIS database for carcinogens. Because risks were characterized for three distinct subpopulations (i.e., adults, children and workers), each of which has different body weights and inhalation rates, adjusted cancer slope factors were calculated for each subpopulation using the following equation from the draft EFH:

$$CSF_{adj} = CSF_{std} \times \left(\frac{BW}{70kg} \right)^{\frac{1}{3}} \quad (7-5)$$

where

- CSF_{adj} = cancer slope factor adjusted to population of interest (per mg/kg-d)
 CSF_{std} = standard cancer slope factor from IRIS (per mg/kg-d)
 BW = body weight for the subpopulation of interest (kg).

Because this adjustment depends on body weight, it was performed anew for each iteration of the Monte Carlo simulation.

7.6.2 Noncarcinogens

For noncarcinogens, the inhalation reference concentrations were applied without adjustment. RfCs are derived using a NOAEL (no observed adverse effect level) or LOAEL (lowest observed adverse effect level) that has been identified from an epidemiological or animal study. To adjust for interspecies differences, the NOAEL or LOAEL must be converted into a human equivalent concentration (HEC) when using animal data. To do so, interspecies differences such as lung tidal volume, breathing frequency, fractional deposition in respiratory tract regions, surface area of respiratory tract region of interest, body weight, and blood:gas (air) partition coefficient must be accounted for. Once a human equivalent concentration for the identified NOAEL or LOAEL has been determined, uncertainty factors are applied. The uncertainty factors account for the use of a LOAEL, interspecies variability, and human variability (e.g., for the protection of sensitive subpopulations, such as children, the elderly, the ill, or those who have been exposed previously).

RfC dosimetry adjustments are based on adult input parameter values. However, an uncertainty factor (usually a factor of 10 or 3) for human variability is intended to account for the uncertainty and variability that might be due to pharmacokinetic and pharmacodynamic considerations, including those related to age differences. For example, airway architecture, breathing patterns and rates, metabolic rates, and sensitivity differ between adults and children, but the application of this uncertainty factor is expected, on a basic level, to include this uncertainty. Therefore, Agency guidance does not recommend additional adjustment of the RfC using ratios of body weights and inhalation rates of children and adults. It would also not be appropriate to convert the RfC to an oral RfD and then modify this value using ratios of body weights and inhalation rates of children and adults. Mechanisms of toxicological action are different for oral exposures, and the same health effect may not occur from the inhalation pathway.

7.7 Calculate Risk or Hazard Quotient

The risk or hazard quotient associated with a unit waste concentration was calculated for each iteration based on the calculated air concentration and the exposure factors selected for the iteration.

Risk for carcinogens was calculated as follows:

$$Risk_{calc'd} = \frac{C_{air} \times 10^{-3} \text{ mg}/\mu\text{g} \times CSF_{adj} \times IR \times ED \times EF}{BW \times AT \times 365 \text{ d/yr}} \quad (7-6)$$

where

$Risk_{calc'd}$ = individual risk associated with unit waste concentration (per mg/kg)

C_{air} = air concentration associated with a unit waste concentration
 ($[\mu\text{g}/\text{m}^3]/[\text{mg}/\text{kg}]$)

CSF_{adj} = cancer slope factor adjusted to population of interest (per mg/kg-d)

IR = inhalation rate (m^3/d)

ED = exposure duration (yr)

EF = exposure frequency (d/yr)

BW = body weight (kg)

AT = averaging time (yr) = 70.

Averaging time is a fixed input to this equation because it must be consistent with the averaging time used to develop the cancer slope factor.

The hazard quotient for noncarcinogens was calculated as follows:

$$HQ_{calc'd} = \frac{C_{air} \times 10^{-3} \text{ mg}/\mu\text{g}}{RfC} \quad (7-7)$$

where

$HQ_{calc'd}$ = hazard quotient associated with unit waste concentration (per mg/kg)

C_{air} = air concentration associated with a unit waste concentration
 ($[\mu\text{g}/\text{m}^3]/[\text{mg}/\text{kg}]$)

RfC = reference concentration (mg/m^3).

Because the hazard quotient equation does not consider exposure factors, there is no difference in results for different receptors at the same location (e.g., adult resident, child resident, and offsite worker). Therefore, only an adult resident and an onsite worker (which differ only in location) were modeled for noncarcinogens. As discussed in Section 7.6.2, the uncertainty factors used in establishing a reference concentration for noncarcinogens should be considered to account for differences between different human receptors.

A single risk or hazard quotient was calculated for adult receptors (adult resident, offsite worker, and onsite worker). However, because the exposure factors for children can vary greatly as the child ages from infancy to adulthood, the child receptor was broken into three age ranges for the purposes of calculating risk. A separate risk or hazard quotient was calculated for each age range for each iteration. For carcinogens, the overall risk to a child resident was the sum of the risks for the three age ranges. For noncarcinogens, it is not appropriate to sum hazard quotients; therefore, the maximum of the three age ranges was used as the overall hazard quotient. Not all the age ranges were used for each iteration. The age at which exposure is assumed to begin was varied for each iteration, and a fixed exposure duration associated with each starting age was then used. Some starting age/exposure duration combinations result in exposure only during a single age range, while others result in exposure over two of the age ranges. None result in exposure over all three age ranges. The values used for starting age and exposure duration for children are detailed in Section 7.5.3.

7.8 Backcalculate Risk-Specific Waste Concentration

The final step in each iteration was to backcalculate the risk-specific waste concentration from the risk or hazard quotient corresponding to a unit waste concentration. Because risk is linear with respect to waste concentration, in the models used in this analysis, this may be done by a simple ratio technique:

$$C_w = \frac{Risk_{crit}}{Risk_{calc'd}} \quad \text{or} \quad \frac{HQ_{crit}}{HQ_{calc'd}} \quad (7-8)$$

where

C_w = risk-specific waste concentration (mg/kg)
 $Risk_{crit}$ = risk criterion (unitless)
 $Risk_{calc'd}$ = risk associated with unit waste concentration (per mg/kg)
 HQ_{crit} = hazard quotient criterion (unitless)
 $HQ_{calc'd}$ = hazard quotient associated with unit waste concentration (per mg/kg).

When a particular constituent had both carcinogenic and noncarcinogenic effects, the carcinogenic risk was used to continue the calculations, because it is generally more conservative.

7.9 Adjustments for Results Not Meeting Linearity Assumption

As mentioned above, risk is assumed to be linear with waste concentration. The assumption of linearity is accurate for the dispersion modeling and the exposure and risk modeling. However, the emissions model is linear only within certain restrictions (e.g., that the concentration does not exceed the saturation concentration). Therefore, it was necessary to check backcalculated results that did not meet those restrictions and modify them. The restrictions on the emissions model, and therefore the checks performed, differ for land-based units and tanks. These are discussed separately in the following sections.

7.9.1 Adjustments for Land-Based Units

For this analysis, wastes were initially assumed to be aqueous phase (i.e., dilute wastes that partition primarily to water within the soil). However, aqueous phase wastes can only occur up to the soil saturation limit. At concentrations above the soil saturation limit, wastes can only occur in organic phase. The soil saturation limit is calculated as follows:

$$C_{sat} = \frac{S}{\rho_b} (K_d \times \rho_b + \theta_w + H' \times \theta_a) \quad (7-9)$$

where

- C_{sat} = soil saturation limit (mg/kg)
- S = solubility limit (mg/L)
- ρ_b = bulk density of soil / waste matrix (kg/L)
- K_d = soil-water partition coefficient (L/kg)
- θ_w = water-filled soil porosity (unitless)
- H' = dimensionless Henry's law constant (unitless, = H/RT)
- θ_a = air-filled soil porosity (unitless).

Wastes can also occur in the organic phase at concentrations below the soil saturation limit, but for most chemicals, the aqueous phase produces greater emissions than the organic phase for the same concentration. A few chemicals (most notably formaldehyde) have greater emissions from the organic phase than the aqueous phase. If the aqueous phase emission rate results in a backcalculated waste concentration that exceeds the soil saturation limit, then the chemical poses no risk in the aqueous phase. However, it may still pose risks in the organic phase at higher concentrations.

In order to address both chemicals with greater emissions from the organic phase and chemicals that pose no risk in the aqueous phase, emissions were also modeled in the organic

phase using a concentration of 1,000,000 mg/kg, or pure component; these were then normalized to a unit concentration by dividing by 1,000,000. When a backcalculated waste concentration based on the aqueous phase assumption exceeded the soil saturation limit, it was recalculated using the normalized organic phase emission rate. Similarly, if the normalized organic phase emission rate was greater than the aqueous phase emission rate, it was used instead. If the backcalculated waste concentration based on the organic phase emission rate exceeds 1,000,000 mg/kg, then the chemical poses no risk in the organic phase.

All results were flagged to indicate the emission rate on which they were based (aqueous or organic). If results were based on the organic phase emission rate, the flag also indicates whether they produced higher emissions and therefore higher risk, or whether there was no risk from the aqueous phase. When there was no risk from either phase, the waste concentration was set to 1,000,000 ppm and flagged as no risk. These flags are very important to note when comparing WMUs and receptors for a particular chemical. As a consequence of the differences in emission rate between the aqueous phase and the organic phase, there can be a significant drop in emission rate (and therefore in risk) right around the soil saturation limit, causing a significant increase in the backcalculated waste concentration from one WMU to another or one receptor to another.

7.9.2 Adjustments for Tanks

For tanks, the concentration limit for the aqueous phase is the solubility of the chemical in water. This was addressed in a manner similar to that described in Section 7.9.1 for land-based units, except that, if the aqueous phase posed no risk and the organic phase did, the waste concentration was capped at the solubility (essentially limiting concentrations to the range that can occur in the aqueous phase.) This was done because it was not considered plausible that pure, organic phase waste would be disposed of directly in tanks.

In addition, for tanks with biodegradation, the biodegradation rates are not linear. At low concentrations, biodegradation is first order. However, at concentrations in excess of the half-saturation level, biodegradation becomes zero order. In order to address this, emissions were modeled in the aqueous phase at the solubility. This emission rate then was normalized to a unit concentration by dividing by the solubility. When the backcalculated waste concentration exceeded the half-saturation constant, suggesting that biodegradation would be zero order, it was recalculated based on the normalized solubility limit emission rate.

All results were flagged to indicate whether they were based on the aqueous emission rate or capped at the solubility and, for tanks with biodegradation, whether emissions were based on first-order or zero-order kinetics. When there was no risk from either aqueous phase or pure component, the waste concentration was set to 1,000,000 mg/L and flagged as no risk. These flags are very important to note when comparing WMUs and receptors for a particular chemical. As a consequence of the differences in emission rate between the aqueous phase and the organic phase, and the difference between first-order and zero-order biodegradation kinetics, there can be a significant drop in emission rate (and therefore in risk) right around the solubility limit or the half-saturation limit, causing a significant increase in the backcalculated waste concentration from one WMU to another or one receptor to another.

7.10 Methodology for Subchronic and Acute Exposure

Exposure and risk modeling for subchronic and acute exposures differed somewhat from the modeling for chronic exposures in several respects. This section describes any differences in the methodology for subchronic and acute exposures for each step of the modeling described in Sections 7.1 through 7.9 for chronic exposures.

7.10.1 Overview

No Monte Carlo analysis was performed for subchronic and acute exposures. Instead, a point estimate of C_w was calculated for each WMU in the Industrial D database. This point estimate is most comparable to the 100th percentile of the distribution generated by the Monte Carlo model for chronic exposures and can be interpreted as the level at which 100 percent of receptors are protected at a particular WMU. A distribution across all WMUs of a specific type was generated from these point estimates, and the 90th percentile of that distribution is what is presented in the results for subchronic and acute exposures.

7.10.2 Select Receptor Location

For subchronic and acute exposures, the most exposed of the 16 receptors at each distance from the site was used. The direction of this receptor varied from WMU to WMU depending on prevailing wind directions.

7.10.3 Obtain Unitized Air Concentration

Unitized air concentrations were interpolated as described in Section 7.3 for chronic exposures, using the maximum UACs at each distance for subchronic and acute averaging times.

7.10.4 Calculate Air Concentration

Air concentration was calculated as described in Section 7.4 (Equation 7-2), using emission rates and residual soil concentrations developed for subchronic and acute averaging times.

7.10.5 Select Exposure Factors

The health benchmarks used for subchronic and acute exposure are analogous to the chronic, noncarcinogen benchmarks used in the chronic exposure analysis. They are expressed as ambient air concentrations, and are compared directly to the modeled air concentration, without the use of exposure factors.

7.10.6 Obtain Health Benchmarks

Health benchmarks for subchronic exposures were obtained for 64 chemicals, and acute health benchmarks were obtained for 35 chemicals, as described in Section 6.

7.10.7 Calculate Hazard Quotient

Hazard quotient was calculated as described in Section 7.7 (Equation 7-7).

7.10.8 Backcalculate Risk-Specific Waste Concentration

Risk-specific waste concentration was backcalculated as described in Section 7.8 (Equation 7-8).

7.10.9 Adjustments for Results Not Meeting Linearity Assumptions

The adjustments described in Section 7.9 were also made for subchronic and acute exposures.

7.11 Modifications to Methodology for Lead

Human health risk assessment for lead is unique. Instead of developing an RfC in the traditional manner, all identified sources of lead exposure (including background) are used to predict blood lead (PbB) levels in the exposed individuals. The predicted PbB levels are compared to a target PbB. PbB levels have long been used as an index of body lead burdens and as an indicator of potential health effects.

The Integrated Exposure Uptake Biokinetic Model (IEUBK) (U.S. EPA, 1994a) was developed to predict PbB levels for an individual child or a population of children. The model was specifically designed to evaluate lead exposure in young children (birth to 7 years of age) because this age group is known to be highly sensitive to lead exposure.

The IEUBK is a versatile risk assessment tool that allows the user to make rapid calculations from a complex array of intake, absorption, distribution, and elimination equations. Default values representing urban background exposures to lead from soil and dust ingestion, air, food, and water are built into the model.

The IEUBK has four primary components: exposure, uptake, biokinetic, and probability distribution. The exposure component integrates media-specific (e.g., air, soil, food, water) lead concentrations and age-dependent media intake rates to calculate age- and media-specific lead intake rates. Thus, the exposure component determines how much lead enters the child's body over the exposure period. The uptake component calculates how much of the lead that is ingested or inhaled is actually absorbed into the blood and the biokinetic component models the distribution of lead from the blood to other body tissues and/or elimination from the body. The final component calculates a plausible probability distribution of PbB for a hypothetical child. The geometric mean PbB is calculated and the probability of exceeding a target PbB is determined.

For this analysis, the IEUBK model was used to identify air concentrations that would result in a probability less than 5 percent of having a PbB level higher than the target PbB and that could be used in place of an RfC in the calculations. Because the IEUBK model cannot be

run in a backcalculation mode, different air concentrations were modeled until one was found that satisfied the 95 percent protection level desired.

Only two receptors were modeled for lead: children ages 0 to 3 years and children ages 3 to 7 years. Adults (including workers) and older children were excluded from the analysis for lead because those age groups are considered less sensitive to lead than 0- to 7-year-olds (and, in fact, the pharmacokinetic relationships in the IEUBK model are only valid for 0- to 7-year-olds).

The IEUBK model inputs are summarized in Table 7-9. These include inhalation rate, body weight, media concentrations (including soil, indoor dust, water, and food), and indoor air concentration as a percentage of outdoor air concentration. The IEUBK model does not support Monte Carlo analysis, so these inputs must be entered as point estimates. To be as consistent as possible with the distributions developed for exposure factors, the median body weight and inhalation rates from the distributions for 0- to 3- and 3- to 10-year-olds were used for 0- to 3- and 3- to 7-year-olds, respectively. Ingestion rate for water was left at the default age-specific values recommended in the IEUBK guidance document (U.S. EPA, 1994a).

Media concentrations were left at the standard background levels. Two background soil concentrations were modeled: 75 mg/kg and 200 mg/kg. These values correspond to the range of typical soil lead levels in urban soil (U.S. EPA, 1994a). Indoor dust concentrations were set to equal soil concentrations and indoor air lead concentrations were equal to outdoor concentrations (the normal default is indoor air = 30 percent of outdoor air). Therefore, it was assumed that children would be exposed 24 hours per day to constant air lead concentrations. This was done for consistency with other constituents for which exposure was assumed to occur 24 h/d with no distinction between indoor and outdoor air made.

Table 7-9. Summary of Inputs for IEUBK Model

Input	Age (yr)	Value	Source
Exposure Factors			
Inhalation rate (m ³ /d)	0-3	6.1	Median of distribution for 0- to 3-yr-olds
	3-7	8.9	Median of distribution for 3- to 10-yr-olds
Body weight (kg)	0-3	12.63	Median of distribution for 0- to 3-yr-olds
	3-7	25.9	Median of distribution for 3- to 10-yr-olds
Media Concentrations			
Soil (µg/g)		75 to 200	U.S.EPA (1994a)
Indoor dust (µg/g)		75 to 200	U.S.EPA (1994a)
Water (µg/L)		4	U.S.EPA (1994a)
Food (µg/d)		varies by age	U.S.EPA (1994a)
Indoor Air Concentration (% of outdoor air conc.)		100%	Assumption/consistency with other constituents

Model outputs were produced for three target blood lead levels (10, 8, and 5 $\mu\text{g/dL}$) and two background soil concentrations (75 and 200 $\mu\text{g/g}$); these are shown in Table 7-10.

Depending on the target PbB and the background soil concentration, acceptable air lead concentrations ranged from background to 3.3 $\mu\text{g/m}^3$. For comparison, the default background air lead concentration used by the IEUBK is 0.1 $\mu\text{g/m}^3$.

For some combinations of target blood lead level, background soil concentration, and age, the background exposures alone led to a probability greater than 5 percent that the blood lead target would be exceeded. This would mean that no exposure to lead in air could be allowed.

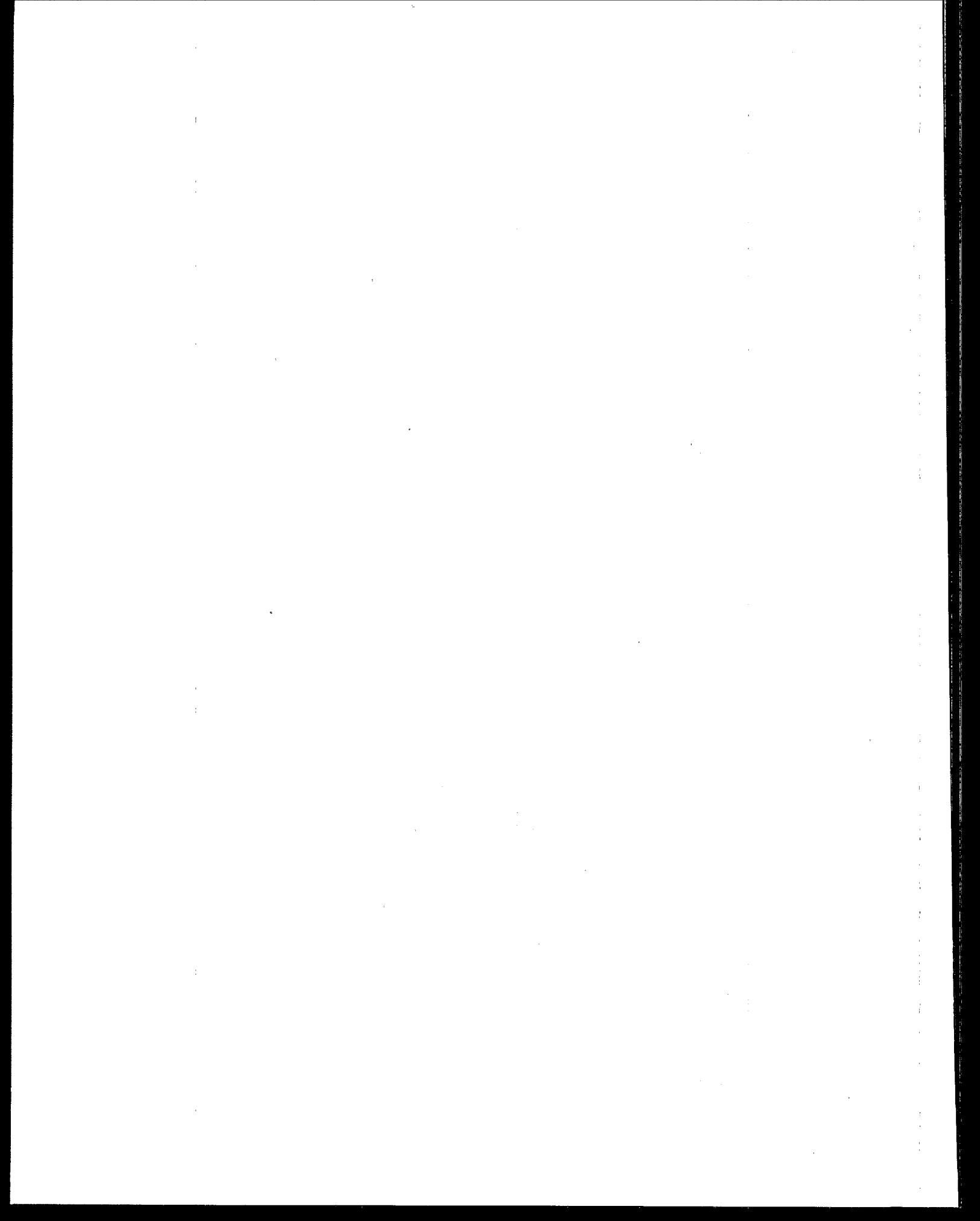
A single air concentration for each age range was needed for use in the Monte Carlo analysis as a surrogate for the RfC. The blood lead level of 10 $\mu\text{g/dL}$ was selected because that level has been identified as a level of concern by the Centers for Disease Control (CDC) (U.S. EPA, 1994a). The IEUBK guidance recommends a background soil concentration of 200 $\mu\text{g/g}$; therefore, that concentration was used. This resulted in air concentrations of 0.5 and 1.8 $\mu\text{g/m}^3$ for the 0- to 3- and 3- to 7-year-old, respectively. These air concentrations were used in the Monte Carlo analysis described above in lieu of a health benchmark.

Table 7-10. Results of IEUBK Modeling

Target Blood Level ($\mu\text{g/dL}$)	Soil/Dust Concentration ($\mu\text{g/g}$)	Air Concentration ^a ($\mu\text{g/m}^3$)	
		0- to 3-year-old	3- to 7-year-old
10	200	0.5	1.8
8	200	0 ^b	0.6
5	200	0 ^b	0 ^b
10	75	2.4	3.3
8	75	1.25	2.1
5	75	0 ^b	0.35

^a Air concentration at which there is a 5% probability of exceeding the target blood lead level.

^b Background exposures alone result in >5% probability of blood lead exceeding target level.



8.0 Analysis of Uncertainty

In any national assessment of this type there are numerous potential uncertainties and a wide range of variability in many of the input parameters. In planning this analysis, it was important to specifically address as much of the variability as possible, either directly in the Monte Carlo analysis or through sensitivity analyses. However, much of the uncertainty could only be addressed qualitatively. The previous sections describe how distributions and point value estimates were made for input parameters and how these were combined through design of the analysis to provide facility-level and national level distributions of C_w . The variability in input parameters that was addressed in this analysis, as well as some significant sources of uncertainty that were not, are discussed in this section according to four broad categories: emissions modeling, dispersion modeling, exposure modeling/risk estimation, and decision rule. This study does not attempt to address modeling error in this analysis directly. Details of the derivation of parameter values based on sensitivity analyses are presented in Appendixes A through D.

Uncertainty introduced into the analysis includes model uncertainty, parameter uncertainty, and parameter variability. The first two, model uncertainty and parameter uncertainty, are generally recognized by risk assessors as major sources of uncertainty.

Parameter uncertainty occurs when parameters in equations cannot be measured precisely and/or accurately either because of equipment limitations or because the quantity being measured varies spatially or temporally. Random, or sample errors, are a common source of parameter uncertainty that is especially critical for small sample sizes. More difficult to recognize are nonrandom or systematic errors that result from bias in sampling, experimental design, or choice of assumptions.

Main Sources of Uncertainty in Risk Assessment

General Type	Specific Source of Uncertainty
Model uncertainty	Surrogate variables
	Excluded variables
	Abnormal conditions
	Incorrect model form
Parameter uncertainty	Measurement errors
	Random errors
	Systematic errors
Parameter variability	Heterogeneity

Model uncertainty is associated with all models used in all phases of a risk assessment. These include the animal models used as surrogates for testing human carcinogenicity, dose-response models used in extrapolations, as well as the computer models used to predict the fate and transport of chemicals in the environment. Computer models are simplifications of reality, requiring exclusion of some variables that influence predictions but cannot be included in models due either to increased complexity or to a lack of data on that parameter. The risk assessor needs to consider the importance of excluded variables on a case-by-case basis because a given variable may be important in some instances and not in others. A similar problem can occur when a model that is applicable under average conditions is used when conditions differ from the average. In addition, choosing the correct model form is often difficult when conflicting theories seem to explain a phenomenon equally well.

Variability, the third source of uncertainty, is often used interchangeably with the term "uncertainty," but this is not strictly correct. Variability may be tied to variations in physical and biological processes and cannot be reduced with additional research or information, although it may be known with greater certainty (e.g., age distribution of a population may be known and represented by the mean age and its standard deviation). "Uncertainty" is a description of the imperfection in knowledge of the true value of a particular parameter or its real variability in an individual or group. In general, uncertainty is reducible by additional information gathering or analysis activities (better data, better models), whereas real variability will not change (although it may be more accurately known) as a result of better or more extensive measurements.

Table 8-1 presents the major categories of uncertainty and how they have been addressed in this study. The columns in the table show model uncertainty, parameter uncertainty, and parameter variability. The rows present the four main model components in the analysis: emissions model, dispersion model, exposure model, and risk model.

8.1 Emissions Modeling

With regard to model error, the CHEMDAT8 model was used for all volatile emissions estimates including acute and subchronic releases and as the basis for estimating soil concentrations that are used in the particulate emission model. As discussed in Section 3.1, there are many features of this model that meet the needs of this analysis. However, the model was developed to address only volatile emissions from these waste management units. Competing mechanisms such as runoff and erosion and leaching are not included in the model. In so much as these competing processes actually occur, the model would tend to slightly overestimate the volatile emissions and waste/soil concentrations in the waste management unit. On the other hand, one could interpret this situation as being representative of WMUs that have leachate controls, such as liners, or erosion and runoff controls. Such controls would tend to inhibit these processes and result in more volatile emissions. Similar lines of argument hold for the calculation of waste/soil concentration that is the basis for estimating particulate emissions of a contaminant.

Among the many parameters needed as inputs to the emissions model, four are critical to the emission estimates: (1) area and depth (or capacity) of the WMU, (2) temperature, (3) volatility, and (4) biodegradation. Both volatility and biodegradation are correlated with

Table 8-1. Summary of How Uncertainties Were Addressed in the Study

	Model Uncertainty	Parameter Uncertainty	Parameter Variability
Emissions Model	Instantaneous release model used for acute and subchronic peak releases	Dependencies of biodegradation, volatility, and temperature addressed through sensitivity analysis and use of seasonal temperature variations	Facility-specific locations, dimensions and waste volumes used to address variability in WMU parameters
Dispersion Model	Model error increased by 2%-10% by not using plume depletion option Acute, subchronic and chronic averages used	Sensitivity analyses conducted on a number of parameters including shape and orientation of WMU, meteorologic data, and receptor grid	29 meteorologic stations used to represent climate regions 14 surface areas used to represent distribution of surface area for landfills, LAUs 7 surface areas and 2 heights used for wastepiles 2 model tanks
Exposure Model	Acute, subchronic and chronic exposures estimated	Sensitivity analysis conducted for receptor grid	16 receptor locations at each distance used at each WMU in Monte Carlo analysis Distributions developed for exposure factors (inhalation rate, body weight, and exposure duration) and used in Monte Carlo analysis
Risk Model	Acute, subchronic and chronic health benchmarks used	Not addressed	Not addressed

temperature and therefore do not vary as independent variables. With regard to area and depth, the data used were derived from facility-specific data contained in the Industrial D database (Shroeder et al., 1987). When more than one WMU of the same type existed at a facility, the combined area of those WMUs was reported in the database. This analysis used the average area of WMUs of a single type when more than one unit existed at the same facility.

In order to address the variability in the surface area and capacity of a waste management unit, unit-specific data were used in the emissions model. The variability in these parameter values spans several orders of magnitude for all WMU types except tanks. Since these are very

sensitive inputs to the emissions model, it was deemed important to capture this variability in the analysis.

The uncertainty associated with the information contained in the Industrial D database is unknown. There are several sources of this uncertainty including (1) missing data on waste volumes or capacity, (2) multiple WMUs of the same type associated with a combined surface area and waste volume, and (3) the accuracy of the reported data (i.e., measurement error). For multiple units of the same type, an average value for the facility was used to model a single WMU. This is not a problematic assumption except for a few facilities with large total surface areas. In such cases, it is unknown whether one large WMU dominates this surface area or the area is evenly distributed across the WMUs.

A sensitivity analysis was conducted to determine the impact of seasonal variations in temperature on both volatility and biodegradation. Section 4 provides a detailed discussion of the assumptions made in modeling emissions from each WMU. For land application units and wastepiles, biodegradation is an important parameter that lowers both emission rates and the average waste/soil concentration. Biodegradation was assumed to occur at temperatures greater than 5°C. In order to turn this on and off, quarterly seasonal temperatures were calculated for each site based on regional-specific meteorologic data. The use of seasonal temperature data was intended to reduce the error in both the biodegradation rate and the temperature-dependent volatilization rate in these land-based WMUs. The landfill scenario assumed no biodegradation occurred and temperature was therefore a less important parameter for this WMU than for the land treatment unit or wastepile. The effect of incorporating seasonal temperature variations in the analysis was chemical-dependent. For most chemicals in the analysis, the difference between using seasonal temperatures or an annual average temperature resulted in little or no change in the overall annual emissions. However, for a few chemicals (see Appendix B) there was a very large impact for certain locations in the United States. These chemicals had rapid biodegradation rates, and turning the biodegradation off for the winter months resulted in significantly higher annual emissions.

Seasonal temperatures were not used to adjust volatility or biodegradation rates for tanks. The assumption in this case is that biologically active tanks must maintain conditions suitable for the biodegradation to occur throughout the year. In addition, the tanks are assumed to have a steady flow-through, and the tank temperature is more a function of the temperature of the entering wastewaters than the ambient temperature.

8.2 Dispersion Modeling

The ISCST3 model was used to calculate the dispersion of particle and gas emissions from a WMU. This model has many capabilities need for this assessment such as the ability to model area, volume, or point sources for chronic, subchronic, and acute averages, and the ability to provide onsite concentrations that were used to evaluate exposure to onsite workers. For dispersion modeling of this type, it is considered a fairly good model, with error within about a factor of 2. It does not include photochemical reactions or degradation of a chemical in the air, which results in additional model uncertainty for some chemicals. In addition, this analysis did not use the plume depletion option. As discussed in Section 5, this option dramatically increases

run time and would have required a much longer schedule to complete. The sensitivity analyses presented in Appendix D show that model error is increased by 2 to 10 percent when plume depletion is turned off.

Dispersion modeling is highly sensitive to meteorological data and the surface area and dimensions of the WMU. Meteorological data used in the dispersion modeling include wind-speed and direction, temperature, precipitation type and amount, and stability class. The ISCST3 model uses hourly data as inputs, and this analysis used 5 years of hourly data to develop chronic (i.e., >1 year), subchronic (i.e., maximum 15 day) and acute (i.e., maximum 1 day) UACs as discussed in Section 5. Dispersion modeling was conducted using meteorological datasets from 29 different meteorological stations around the continental United States. This provided regional representation of the variability in meteorological data. These datasets were combined with 14 surface areas representing the distribution for landfills and land treatment units. Combined, this provides 406 different sets of UACs (a set consists of all UACs at various distances and directions at one location) to use with emissions data to estimate air concentrations. Similarly, wastepiles used 406 different sets of UACs based on 7 surface areas, 2 heights, and 29 meteorological stations. Tanks used the same 29 meteorological stations and two different characterizations of tanks, providing 58 sets of UACs. To minimize error associated with the use of discrete surface areas, an interpolation routine was used to estimate the UAC for a specific surface area, using the UACs for the areas immediately above and below the WMU used.

Obviously, 29 meteorological stations do not represent every site-specific condition that could exist in the continental United States. However, based on the EQM report (1993), it is believed that these stations provide a representation of the variability in wind rose for the U.S.-climate regions. Quantifying the error associated with the use of this limited number of meteorological datasets would require comparison of results with more site-specific data, which has not been done. However, a detailed analysis of the wind rose for the 29 met stations is presented in Appendix B.

**Shape of Wind Rose for
29 Meteorological Stations**

Shape of Wind Rose	No. of Stations
Narrowly distributed	10
Moderately distributed	4
Evenly distributed	6
Bimodally distributed	9

8.3 Exposure Modeling/Risk Estimation

The potential location of receptors was the main variability addressed in the exposure modeling. The data used to identify and characterize WMUs contained no information on the location and types of receptors near the facility. Many previous risk analyses have used the maximum point of exposure at some prespecified distance from the WMU as the point for analysis. Such an approach is usually criticized as being overly conservative because it does not consider the possibility of no one living at that exact point. Since individuals may potentially be located in any direction and at various distances from a facility, this analysis developed an explicit way to incorporate this consideration. First, a sensitivity analysis was conducted to

determine a reasonable distance at which to bound the analysis. This sensitivity analysis showed that, beyond 1,000 meters, most air concentrations are a small percentage (less than 10 percent) of the concentration at the point of maximum exposure. Therefore, 1,000 meters was used as the outer bound on the distance of receptors included in this analysis. Next, a receptor grid was set up to allow individuals to reside in any of 16 directions and at distances of 25, 50, 75, 150, 500, and 1,000 meters from the edge of the unit. The Monte Carlo analysis uses a uniform distribution that gives equal probability to a receptor being located in any of 16 directions at each distance.

The exposure factors used in the analysis, including inhalation rate, body weight, and duration of exposure, are quite variable. To include this variability explicitly in the analysis, distributions for these variables were used for each receptor in the analysis. Thus, for each Monte Carlo realization for each receptor type, values for each of these variables were selected from the specified distribution.

At each WMU modeled, a distribution of C_w was developed that incorporated the uncertainty in the location of the receptor and the variability in the exposure factors used to characterize the receptor. Note that the Monte Carlo analysis for noncarcinogens incorporates uncertainty only about receptor location since exposure factors are not used in the calculation.

Risk estimation is a straightforward calculation using health benchmarks. There is recognized uncertainty in the health benchmarks; however, this uncertainty has not been explicitly addressed in this analysis.

The overall output of the analysis includes specific consideration of the variability in site-specific WMU information, regional-specific meteorological conditions, the location of receptors, and the exposure factors for each receptor to provide national distributions of a risk-specific waste concentration across all facilities of a specified type. These distributions are presented in Volume III.

8.4 Indirect Exposures

This risk analysis addresses only inhalation exposure to humans. This is considered a direct exposure pathway. Indirect exposures include exposure pathways such as contaminated produce or meats due to the uptake and bioaccumulation of contaminants in the food chain. Although this analysis focused only on releases of the contaminants to air, it is possible that these contaminants could accumulate in plants through air-to-plant transfer mechanisms or by direct deposition onto plants. Animals could then come into contact with contaminated plants, or even contaminated soil, and accumulate the contaminant in their bodies (bioaccumulation). Because of the short time frame the project was to be completed in and the complexity associated with modeling indirect risks, the consent decree with EDF did not require the Agency to evaluate this pathway.

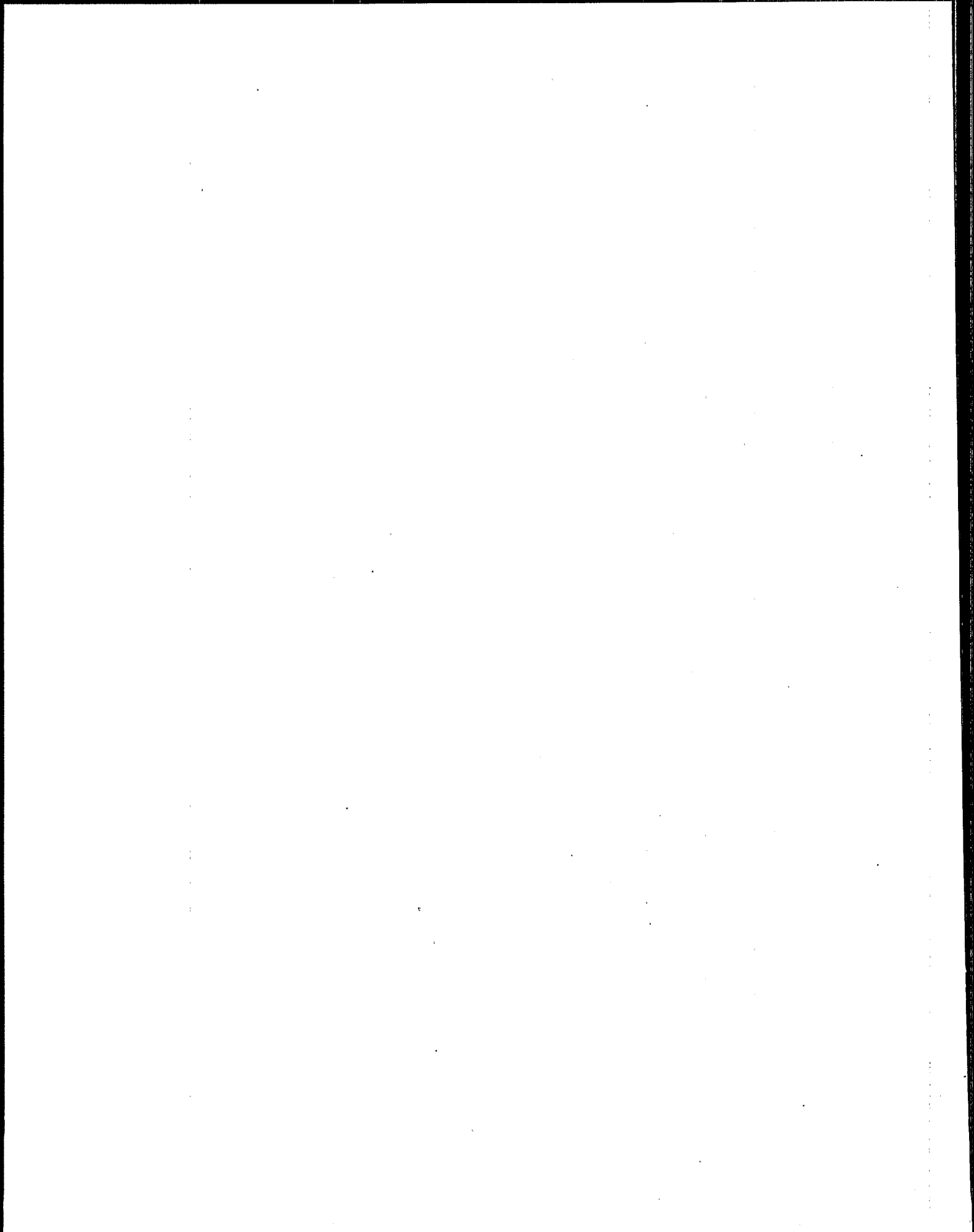
EPA conducted a preliminary analysis to determine if any constituents included in this analysis have the potential to be taken up by plants and accumulated to the point of presenting a

potential concern for humans or domestic animals such as beef or dairy cows that might graze in fields adjacent to a WMU.

In summary, EPA looked at a waste stream in which polychlorinated aromatic hydrocarbons (PAHs) were the constituents of concern from a risk perspective. Releases from an LAU were modeled with receptors located randomly between 75 and 1,000 m. The concentration of constituents in contaminated produce could be attributed to three mechanisms—vapor transfers, direct deposition from air, and uptake from soil, with vapor transfers being the predominant mechanism of contamination. The risk from ingestion of contaminated produce as a percent of total risk from all ingestion pathways is quite significant for the farmer scenario and less significant for the home gardener.

Two important points need to be made regarding this modeling exercise. First, the vapor transfer coefficients used for PAHs are very high and thought to have a high level of uncertainty. Studies are currently underway to address this uncertainty but the results of such studies are not available at this time. One criticism is that these vapor transfer coefficients greatly overestimate the concentration of PAHs in plants and thus lead to overestimates of risk. Second, the farmer scenario has much higher risk from homegrown produce than the home gardener due to differences in ingestion rates for these two groups. At this time, it is not known which scenario would be the most appropriate to use for the air characteristic analysis or the prevalence of farmers or home gardeners in the vicinity of these types of facilities.

Although this sensitivity analysis suggests that indirect pathways may be important to consider in the air characteristic risk analysis, this analysis is very limited in terms of constituents, WMUs, settings and scenarios. In a sense, this sensitivity analysis should be considered a bounding analysis since it is based on a set of inputs that is biased toward determining if the air-to-plant pathway could be a concern for any of the constituents.



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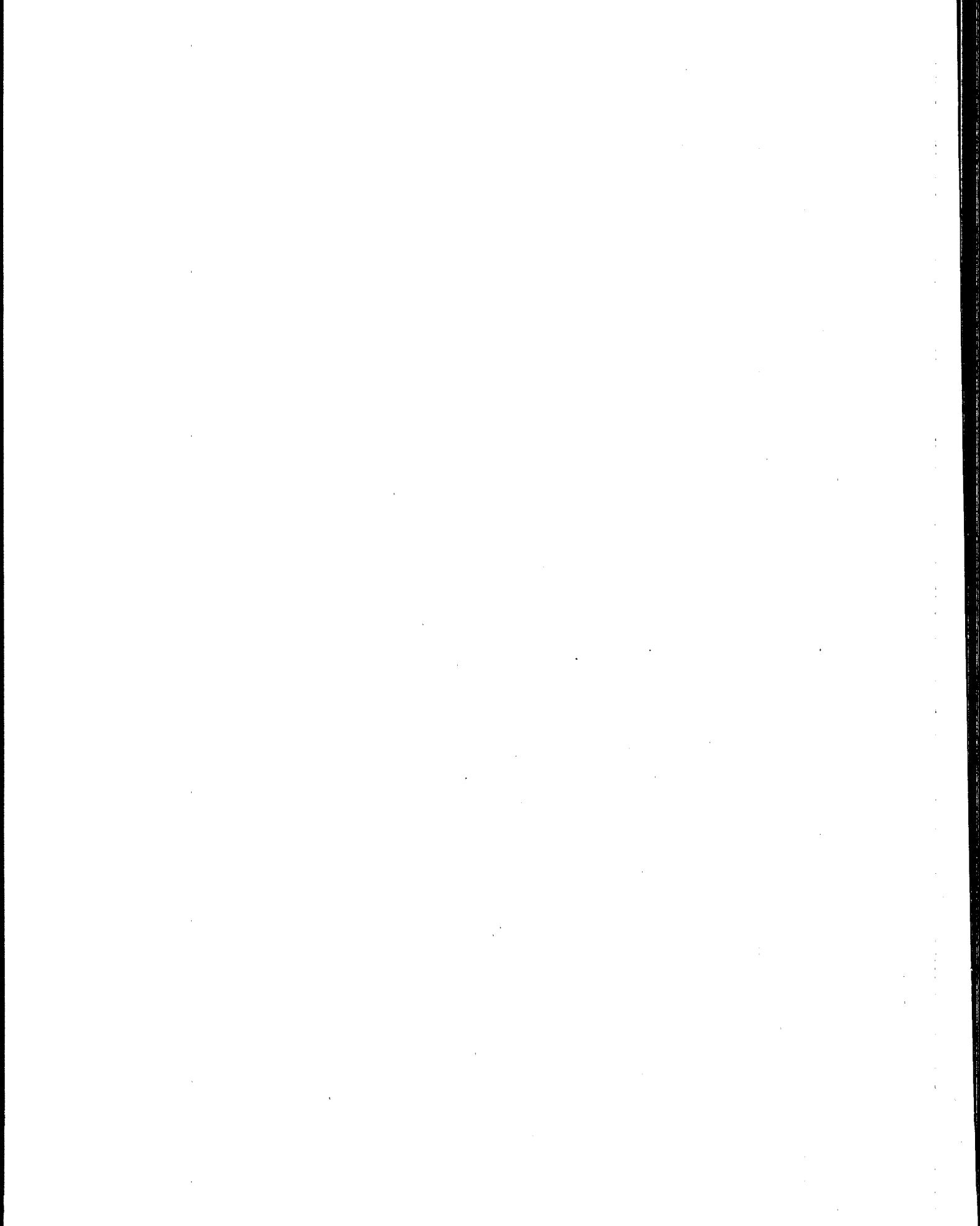
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Appendix A

Basic Dalenius-Hodges Procedure for Constructing Strata



A. Basic Dalenius-Hodges Procedure for Constructing Strata (applies to census data)

1. Select an available variable X correlated with the variable Y of interest. (Both X and Y are assumed to be continuous.)
2. Form a relative frequency histogram of X with respect to M prespecified intervals with breakpoints $a_0, a_1, a_2, \dots, a_M$. Let f_i denote the frequency count for the i^{th} interval. (Note that M should be fairly large, e.g., at least 50 and preferably 100, since the ultimate strata will be formed as the union of a subset of contiguous intervals. The intervals do not have to be the same length but it may be convenient to use one or two standard lengths.)
3. For each interval i ($i=1,2,\dots,M$), compute $Z_i = \sqrt{L_i f_i}$ where $L_i = a_i - a_{i-1}$ is the length of the i^{th} interval.
4. Compute $CUMZ_i = \sum_{j=1}^i Z_j$.
5. Determine stratum breakpoints by breaking $CUMZ$ into equal intervals of length $Q = CUMZ_M/H$, where H is the desired number of strata (e.g., if $H=5$, then stratum boundaries would be formed at $Q, 2Q, 3Q$, and $4Q$).
6. Sequentially cluster the intervals into strata in order to redefine breakpoints in terms of X variable.
7. Select random samples of size n within each stratum.

In practice, H is normally about 4, 5, or 6. This procedure produces a sample that is near optimal for estimating the population mean of Y if X and Y are highly correlated.

Modification of the Basic Procedure for the Current Context

At Step 2, determine f_i as the sum of the weights of the units falling into the i^{th} interval.¹ At Step 7, select a unit (or facility, depending on whether X and associated weights relate to units or facilities) near the midpoint of each stratum as the "representative" unit. Its percentile in the population of units would be estimated as (sum of weights for all units having $X_s \leq X$ of given unit)/(sum of all weights).

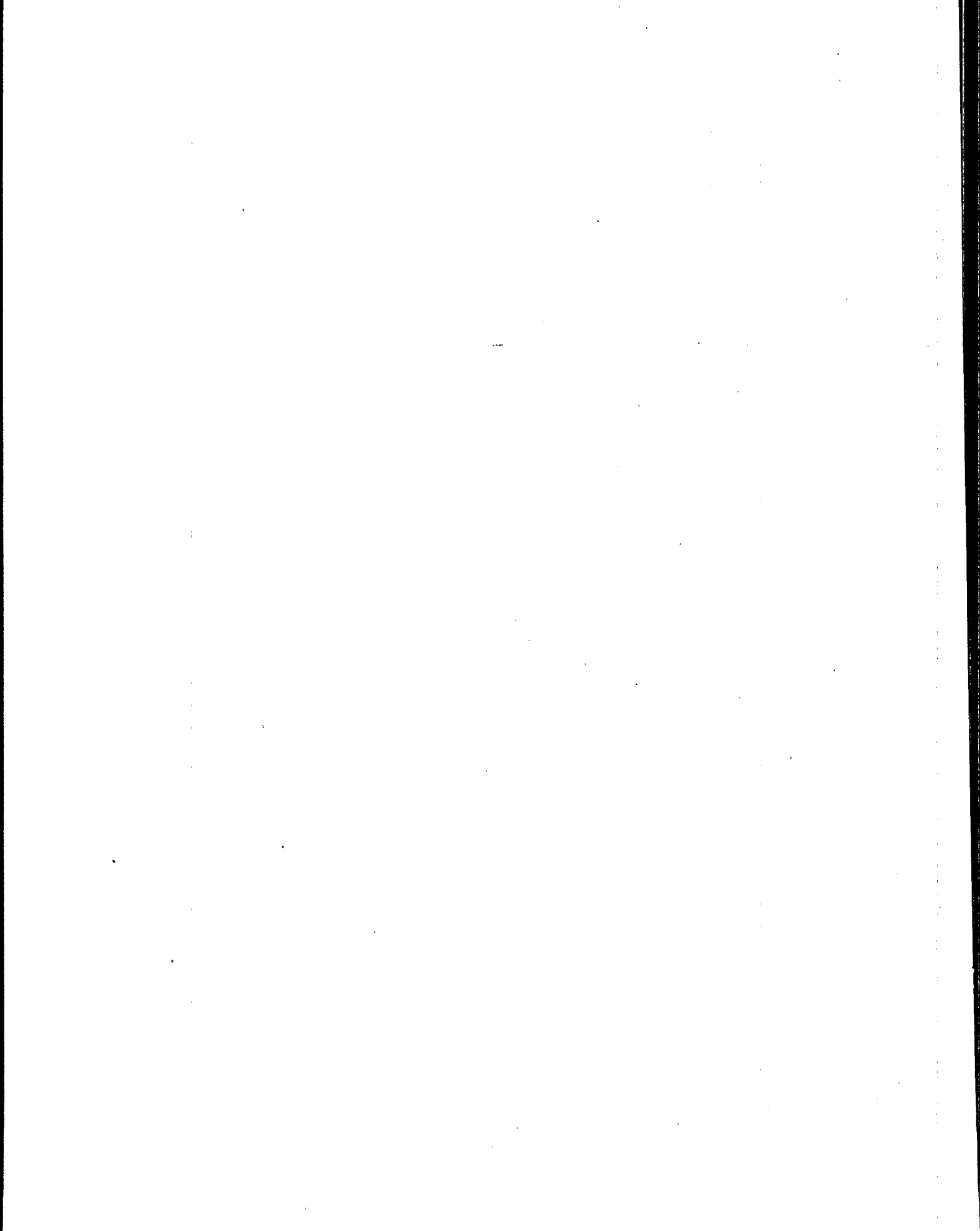
¹ Note that the current weights relate to the population of facilities; to get weights appropriate to units (which one should do if x is defined as a unit-specific measure rather than a facility-specific measure), one should multiply the number of units at a facility by the facility's weight. As a check, the sum of the current weights should produce an estimate of the number of facilities in the population and the sum of the (weight \times number of units) should produce an estimate of the number of units in the population.

The above procedure was applied to distributions for landfills (LFs), land application units (LAUs), surface impoundments (SIs), and wastepiles (WPs). It was also applied to the combined LF/LAU/SI distribution. In each case, the procedure yields a good characterization of the right tail of the distribution, but a poor characterization of the lower portion of the distribution. As a result, the procedure was modified by collapsing two or more of the highest strata into one and successively splitting the lowest stratum into two or more strata. The splitting was performed by choosing stratum breakpoints within the lowest initial at $CUMZ < Q$ (boundary of the initial stratum), $CUMZ < Q/2$, $CUMZ < Q/4$, $CUMZ < Q/8$, etc.

NOTE: If facility weights are used, but X =average unit area, then the distribution being characterized is the facility distribution of average unit areas. A percentile, X_p , of this distribution has the following interpretation: 100P% of the facilities have average unit size $< X_p$. On the other hand, if unit level weights are used, then the P^{th} percentile, X_p , is interpreted as: 100P% of the units have size $< X_p$. The latter is probably the most meaningful, but it can only be approximated since within-facility variation in unit sizes is not available from the survey data.

Appendix B

Chemical-Specific Data



B. Chemical-Specific Data

Key chemical-specific input parameters include: air-liquid equilibrium partitioning coefficient (vapor pressure or Henry's law constant), liquid-solid equilibrium partitioning coefficient (log octanol-water partition coefficient for organics), biodegradation rate constants, and liquid and air diffusivities. The HWIR chemical properties database (RTI, 1995) was used as the primary data source for the physical and chemical properties for the constituents being modeled. This chemical properties database provided the following chemical-specific input parameters: molecular weight, vapor pressure, Henry's law constant, solubility, liquid and air diffusivities, log octanol-water partition coefficient, and the soil biodegradation rate constants. The CHEMDAT8 chemical properties database (U.S. EPA, 1994) was used as a secondary data source for the physical and chemical properties not included in the HWIR data base. The CHEMDAT8 chemical properties database primarily provided the following chemical-specific input parameters: density, boiling point, Antoine's coefficients (for adjusting vapor pressure to temperature), and biodegradation rate constants for tanks. Hydrolysis rates were taken from Kollig et al. (1993). The biodegradation rate constants in the downloaded CHEMDAT8 data base file were compared with the values reported in the summary report that provided the basis for the CHEMDAT8 tank biodegradation rate values (Coburn et al., 1988). Tank biodegradation rates constants for compounds with no data were assigned biodegradation rates equal to the most similar compound in the biodegradation rate data base (or set to zero for metals). The chemical specific input parameters used for the emission model estimates are presented in Table B-1.

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TABLE B-1. Chemical Specific Input Parameters

CAS #	COMPOUND NAME	Mol. Wt.	Density	VAP. Press.	H Law Const.	Diffusivity In Water	Diffusivity In Air	Antolnes' Vapor Pressure Coefficients			log Oct Water Part. Coeff.	Kmax	K1	Hydrol. Rate	Soil Biodeg. Rate	Solubility
		(g/mol)	(g/cc)	(mmHg)	(atm-m3/mol)	(cm2/sec)	(cm2/sec)	A	B	C		hr.	L/g-hr.	sec-1	sec-1	mg/L
50000	Formaldehyde	30.03	0.97	5240	3.4E-07	1.98E-05	1.78E-01	7.195	971	244	-0.05	5	0.25	0	6.08E-10	5.50E+05
50328	Benzo(a)pyrene	252.32	1.11	5.5E-09	1.1E-06	9.00E-06	4.30E-02	9.246	3724	273	6.11	0.001	0.31	0	4.61E-08	2.50E-02
55185	N-Nitrosodiethylamine	102.14		0.86	3.6E-06	8.00E-06	8.00E-02			273	0.48	4.4	0.45		1.56E-08	9.30E+04
56235	Carbon tetrachloride	153.82	1.59	115	0.0304	8.80E-06	7.80E-02	6.934	1242	230	2.73	1.5	1.50	0	3.13E-08	7.93E+02
56495	3-Methylcholanthrene	268.36	1.02	7.7E-09	9.4E-07	5.36E-06	2.09E-02	8.164	3364	273	6.42	0.001	0.31	0	1.22E-07	3.23E-03
57976	7,12-Dimethylbenz[a]anthracene	256.35	1.02	5.6E-09	3.1E-08	4.98E-06	4.61E-02	6.955	2163	171	6.62	0.001	0.31	0	2.43E-09	2.50E-02
62533	Aniline	93.13	1.02	0.49	1.9E-06	8.30E-06	7.00E-02	6.950	1467	177	0.98	7.1	21.00	0	6.95E-10	3.61E+04
67561	Methanol	32.04	0.79	126	4.6E-06	1.64E-05	1.50E-01	7.897	1474	229	-0.71	18	0.20	0	6.08E-10	1.00E+06
67641	Acetone	58.08	0.79	230	3.9E-05	1.14E-05	1.24E-01	7.117	1211	230	-0.24	1.3	1.15	0	6.08E-10	1.00E+06
67663	Chloroform	119.38	1.49	197	0.00367	1.00E-05	1.04E-01	6.493	929	196	1.92	28	0.79	0	2.43E-09	7.92E+03
67721	Hexachloroethane	236.74	2.09	0.21	0.00389	6.80E-06	2.49E-03	7.228	1348	133	4	0.001	0.03	0	1.56E-08	5.00E+01
68122	N,N-Dimethyl formamide	73.09	0.9445	4	1.9E-07	1.92E-05	9.39E-02	6.928	1401	196	-1.01	9.7	0.13	0	1.00E-20	1.00E+06
71432	Benzene	78.11	0.87	95	0.00558	9.80E-06	8.80E-02	6.905	1211	221	2.13	19	1.40	0	1.39E-09	1.75E+03
71556	1,1,1-Trichloroethane	133.4	1.33	124	0.0172	8.80E-06	7.80E-02	6.827	1147	219	2.48	3.5	0.74	2E-08	2.37E-08	1.33E+03
74839	Methyl bromide	94.94	1.41	1620	0.00624	1.21E-05	7.28E-02	7.566	1301	273	1.19	10.76	0.35	0	2.43E-09	1.52E+04
74873	Methyl chloride	50.49	0.95	4300	0.00882	6.50E-06	1.26E-01	7.093	949	249	0.91	10.76	0.72	0	2.43E-09	5.33E+03
75014	Vinyl chloride	62.5	0.91	2980	0.027	1.23E-05	1.06E-01	6.991	969	251	1.5	10.76	0.14	0	1.56E-08	2.76E+03
75058	Acetonitrile	41.05	0.78	91.1	3.5E-05	1.66E-05	1.28E-01	7.119	1314	230	-0.34	9.7	0.10	0	2.43E-09	1.00E+06
75070	Acetaldehyde	44.05	0.788	902	7.9E-05	1.41E-05	1.24E-01	8.005	1600	292	1.25	82.42	0.20	0	1.00E-20	1.00E+06
75092	Methylene chloride	84.93	1.34	433	0.00219	1.17E-05	1.01E-01	6.968	1074	223	1.25	18	0.38	0	2.43E-09	1.30E+04
75150	Carbon disulfide	76.14	1.26	359	0.03022	1.00E-05	1.04E-01	6.942	1169	242	2	15.3	0.89	0	1.00E-20	1.19E+03
75218	Ethylene oxide	44.06	0.87	1094	0.00012	1.45E-05	1.04E-01	7.128	1055	238	-0.3	4.2	0.91	0	1.00E-20	3.83E+05
75252	Tribromomethane	252.73	2.89	5.51	0.00054	1.03E-05	1.49E-02	7.988	2159	273	2.35	10.76	1.01	0	1.56E-08	3.10E+03
75274	Bromodichloromethane	163.83	1.97	50	0.0016	1.06E-05	2.98E-02	7.966	1847	273	2.1	10.76	0.70	0	1.00E-20	6.74E+03
75354	1,1-Dichloroethylene	96.94	1.213	600	0.0261	1.04E-05	9.00E-02	6.972	1099	237	2.13	10.76	0.90	0	1.56E-08	2.25E+03
75569	Propylene oxide	58.08	0.83	532.1	8.5E-05	1.00E-05	1.04E-01	7.067	1133	236	0.03	17.56	0.17	0	1.00E-20	4.76E+05
75694	Trichlorofluoromethane	137.37	1.49	803	0.097	9.70E-06	8.70E-02	6.884	1043	237	2.53	1.076	0.12	0	3.13E-08	1.10E+03
75718	Dichlorodifluoromethane	120.91	1.41	4850	0.343	8.00E-06	8.00E-02	7.590	1329	273	2.16	1.076	0.07	0	1.56E-08	2.80E+02
76131	1,1,2-Trichloro-1,2,2-trifluoroethane	187.38	1.41	332	0.4815	8.20E-06	7.80E-02	8.784	1894	273	3.16	0.001	0.03	0	1.00E-20	1.70E+02
77474	Hexachlorocyclopentadiene	272.77	1.7	0.0596	0.027	6.16E-06	5.61E-02	8.415	2835	273	5.39	0.001	0.03	0	2.43E-09	1.80E+00
78591	Isophorone	138.21	0.92	0.438	6.6E-06	6.76E-06	6.23E-02	7.963	2481	273	1.7	15.3	0.60	0	2.43E-09	1.20E+04
78875	1,2-Dichloropropane	112.99	1.156	52	0.0028	8.73E-06	7.82E-02	6.980	1380	223	1.97	17	1.40	0	1.12E-07	2.80E+03
78933	Methyl ethyl ketone	72.11	0.82	95.3	5.6E-05	9.80E-06	8.08E-02	7.112	1305	229	0.28	2	0.20	0	6.08E-10	2.23E+05
79005	1,1,2-Trichloroethane	133.4	1.435	23.3	0.00091	8.80E-06	7.80E-02	7.192	1480	229	2.05	3.5	0.74	0	3.17E-08	4.42E+03
79016	Trichloroethylene	131.39	1.4	73.5	0.0103	9.10E-06	7.90E-02	6.518	1019	193	2.71	3.9	0.88	0	3.13E-08	1.10E+03
79061	Acrylamide	71.08	0.84	0.007	1E-09	1.06E-05	9.70E-02	11.293	3940	273	-0.96	9.7	0.27	0	6.52E-11	6.40E+05
79107	Acrylic acid	72.06	1.12	4	1.2E-07	1.06E-05	9.60E-02	5.652	649	155	0.35	17.56	0.18	0	1.00E-20	1.00E+06

TABLE B-1. (continued)

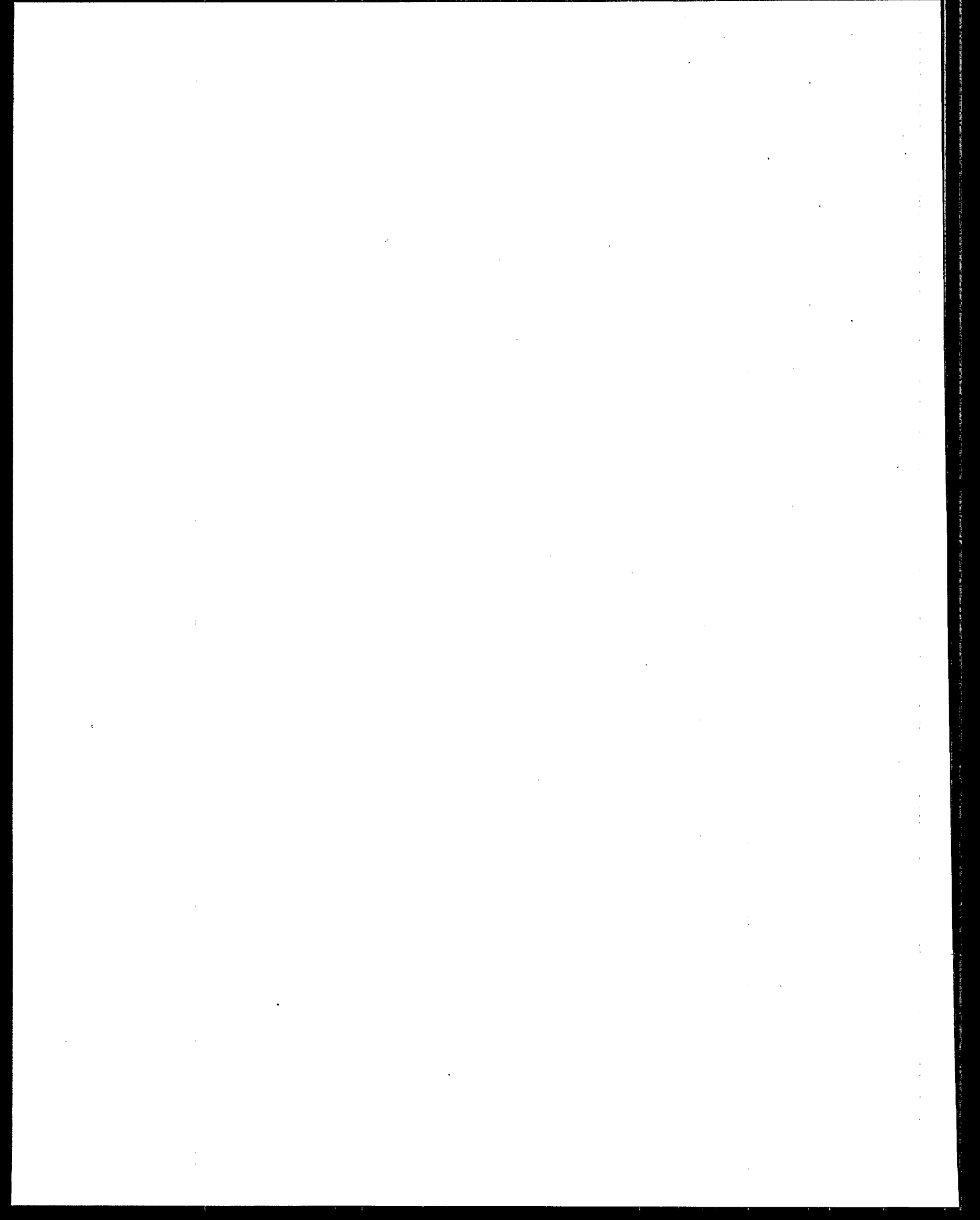
CAS #	COMPOUND NAME	Mol. Wt.	Density	VAP. Press.	H Law Const.	Diffusivity in Water	Diffusivity in Air	Antoinnes' Vapor Pressure Coefficients			log Oct Water	Kmax	K1	Hydrol. Rate	Soil Biodeg. Rate	Solubility
		(g/mol)	(g/cc)	(mmHg)	(atm-m3/mol)	(cm2/sec)	(cm2/sec)	A	B	C	Part. Coeff.	mgVO/g-hr.	L/g-hr.	sec-1	sec-1	mg/L
79345	1,1,2,2-Tetrachloroethane	167.85	1.59	4.62	0.00035	7.90E-06	7.10E-02	6.894	1355	192	2.39	6.2	0.68	0	3.82E-09	2.97E+03
79469	2-Nitropropane	89.09	0.9876	18	0.00012	1.01E-05	9.23E-02	7.272	1531	229	0.87	9.7	0.42	0	1.56E-08	1.70E+04
80626	Methyl methacrylate	100.12	0.95	38.4	0.00034	8.60E-06	7.70E-02	6.517	1052	188	1.38	17.56	4.30	0	2.43E-09	1.50E+04
85449	Phthalic anhydride	148.12	1.33	0.00052	1.6E-08	9.60E-06	7.10E-02	8.022	2869	273	-0.62	17.56	0.08	0.0155	1.00E-20	6.20E+03
87683	Hexachloro-1,3-butadiene	260.76	1.67	0.221	0.00815	6.16E-06	5.61E-02	7.485	1956	215	4.81	0.001	0.03	0	1.56E-08	3.23E+00
91203	Naphthalene	128.17	1.14	0.085	0.00048	7.50E-06	5.90E-02	7.373	1968	223	3.36	42.47	1.00	0	4.17E-09	3.10E+01
92875	Benzidine	184.24	1.02	8E-09	3.9E-11	1.50E-05	8.00E-02	7.542	2626	163	1.66	31.1	0.66	0	6.95E-10	5.00E+02
95501	o-Dichlorobenzene	147	1.31	1.36	0.0019	7.90E-06	6.90E-02	6.883	1538	205	3.43	2.5	0.58	0	1.56E-08	1.56E+02
95534	o-Toluidine	107.16	0.989	0.32	2.7E-06	9.12E-06	7.14E-02	7.197	1683	191	1.34	31.1	0.86	0	6.08E-10	1.66E+04
95578	2-Chlorophenol	128.56	1.26	2.34	0.00039	9.46E-06	5.01E-02	6.877	1472	193	2.15	15	0.89	0	1.00E-20	2.20E+04
95658	3,4-Dimethylphenol	122.17	0.97	0.05836	2.3E-07	8.33E-06	6.02E-02	7.504	1940	197	2.23	5.5	1.05	0	1.00E-20	4.00E+04
96128	1,2-Dibromo-3-chloropropane	236.33	1.41	0.58	0.00015	7.02E-06	2.12E-02	8.073	2436	273	2.34	10.76	0.16	0	1.56E-08	1.23E+03
98011	Furfural	96.09	1.16	2.21	4E-06	1.04E-05	6.72E-02	6.575	1199	163	0.41	17.56	0.54	0	1.00E-20	1.10E+05
98828	Cumene	120.19	0.86	4.5	1.16	7.10E-06	8.60E-02	6.963	1461	208	3.58	31.1	2.88	0	6.95E-10	6.13E+01
98953	Nitrobenzene	123.11	1.2	0.245	2.4E-05	8.60E-06	7.60E-02	7.115	1747	202	1.84	11	2.30	0	1.71E-08	2.09E+03
100414	Ethylbenzene	106.17	0.87	9.6	0.00788	7.80E-06	7.50E-02	6.975	1424	213	3.14	6.8	2.10	0	8.69E-10	1.69E+02
100425	Styrene	104.15	0.9	6.12	0.00275	8.00E-06	7.10E-02	6.945	1437	208	2.94	31.1	0.11	0	2.43E-09	3.10E+02
106467	p-Dichlorobenzene	147	1.29	1	0.0024	7.90E-06	6.90E-02	7.199	1690	218	3.42	6.4	2.30	0	1.00E-20	7.38E+01
106887	1,2-Epoxybutane	72.11	0.826	207.912	0.00046	1.03E-05	1.35E-01	6.832	1141	228	1.441	10.76	0.48	0	1.00E-20	4.28E+04
106898	Epichlorohydrin	92.53	1.18	16.4	3E-05	9.80E-06	8.60E-02	8.229	2087	273	0.25	10.76	0.14	0	2.43E-09	6.59E+04
106934	Ethylene dibromide	187.86	2.7	13.3	0.00074	1.19E-05	2.17E-02	7.345	1675	245	1.96	10.76	0.55	0	1.56E-08	4.18E+03
106990	1,3-Butadiene	54.09	0.76	2110	0.0736	1.08E-05	2.49E-01	7.217	1145	269	1.99	15.3	0.69	0	1.00E-20	7.35E+02
107028	Acrolein	56.06	0.84	274	0.00012	1.22E-05	1.05E-01	7.213	1297	247	-0.01	7.8	0.34	0	2.43E-09	2.13E+05
107051	Allyl chloride	76.53	0.94	368	0.011	1.08E-05	1.17E-01	7.576	1494	273	1.45	10.76	0.31	0	1.21E-09	3.37E+03
107062	1,2-Dichloroethane	98.96	1.26	78.9	0.00098	9.90E-06	1.04E-01	7.068	1293	225	1.47	2.1	0.98	0	1.56E-08	8.52E+03
107131	Acrylonitrile	53.06	0.97	109	0.0001	1.34E-05	1.22E-01	7.110	1336	238	0.25	18	0.75	0	2.00E-09	7.40E+04
107211	Ethylene glycol	62.07	1.11	0.092	6E-08	1.22E-05	1.08E-01	8.091	2089	204	-1.36	17.56	0.06	0	1.00E-20	1.00E+06
108054	Vinyl acetate	86.09	0.93	90.2	0.00051	9.20E-06	8.50E-02	7.210	1296	227	0.73	17.56	0.30	0	1.00E-20	2.00E+04
108101	Methyl isobutyl ketone	100.16	0.8	19.9	0.00014	7.80E-06	7.50E-02	6.672	1168	192	1.19	0.74	0.45	0	6.08E-10	1.90E+04
108883	Toluene	92.14	0.87	28.4	0.00664	8.60E-06	8.70E-02	6.954	1345	219	2.75	6.7	2.40	0	1.91E-09	5.26E+02
108907	Chlorobenzene	112.56	1.11	12	0.0037	8.70E-06	7.30E-02	6.978	1431	218	2.86	0.39	10.00	0	1.30E-08	4.72E+02
108930	Cyclohexanol	100.2	0.95	1.22	4.5E-06	8.31E-06	2.14E-01	6.255	913	109	1.577	17.56	0.54	0	1.00E-20	3.60E+04
108952	Phenol	94.11	1.07	0.276	4E-07	9.10E-06	8.20E-02	7.133	1517	175	1.48	97	13.00	0	8.69E-10	8.28E+04
109864	2-Methoxyethanol	76.09		2.55697	2.6E-07	8.00E-06	8.00E-02			273	-0.77	19.8	1.00		1.00E-20	1.00E+06
110496	2-Methoxyethanol acetate	130.15		9.28503	1.6E-06	8.00E-06	8.00E-02			273	0	19.8	1.00		1.00E-20	1.00E+06
110543	n-Hexane	86.18	0.66	151	0.0143	7.77E-06	2.00E-01	6.876	1171	224	4	15.3	1.47	0	1.00E-20	1.24E+01
110805	2-Ethoxyethanol	90.12	0.9	5.31	3.5E-07	9.57E-06	9.47E-02	7.874	1844	234	-0.1	19.8	1.00	0	2.43E-09	1.00E+06
110861	Pyridine	79.1	0.98	20.8	8.9E-06	7.60E-06	9.10E-02	7.041	1374	215	0.67	35.03	0.24	0	6.08E-10	1.00E+06

TABLE B-1. (continued)

CAS #	COMPOUND NAME	Mol. Wt. (g/mol)	Density (g/cc)	VAP. Press. (mmHg)	H Law Const. (atm-m ³ /mol)	Diffusivity In Water (cm ² /sec)	Diffusivity In Air (cm ² /sec)	Antolnes' Vapor Pressure Coefficients			log Oct Water Part. Coeff.	Kmax mgVO/g-hr.	K1 L/g-hr.	Hydrol. Rate sec-1	Soil Biodeg. Rate sec-1	Solubility mg/L
								A	B	C						
111159	2-Ethoxyethanol acetate	143.01		11.6912	2.2E-06	8.00E-06	8.00E-02			273	0	19.8	1.00		1.00E-20	1.00E+06
118741	Hexachlorobenzene	284.78	2.04	1.8E-05	0.00132	5.91E-06	5.42E-02	9.554	3249	203	5.89	0.001	0.03	0	1.82E-07	6.20E+00
120821	1,2,4-Trichlorobenzene	181.45	1.41	0.431	0.00142	8.23E-06	3.00E-02	7.706	2243	253	4.01	1.076	0.44	0	1.56E-08	3.00E+02
121142	2,4-Dinitrotoluene	182.14	1.31	0.00015	9.3E-08	7.06E-06	2.03E-01	7.981	3074	280	2.01	9.7	0.78	0	1.56E-08	2.70E+02
121448	Triethylamine	101.19	0.7326	57.07	0.00014	7.88E-06	8.81E-02	6.959	1272	223	1.45	9.7	1.06	0	1.00E-20	5.50E+04
122667	1,2-Diphenylhydrazine	184.24	1.19	0.00043	1.5E-06	7.36E-06	3.17E-02	13.836	5403	273	2.94	19	1.91	0	1.00E-20	6.80E+01
123911	1,4-Dioxane	88.11	1.03	38.1	4.8E-06	1.02E-05	2.29E-01	7.351	1518	238	-0.39	17.56	0.39	0	1.56E-08	1.00E+06
124481	Chlorodibromomethane	208.28	2.451	4.9	0.00078	1.05E-05	1.96E-02	8.220	2100	273	2.17	10.76	0.04	0	1.56E-08	2.60E+03
126998	Chloroprene	88.54	0.958	213.658	0.0143	1.00E-05	1.04E-01	6.161	783	180	2.08	10.76	0.22	0	1.56E-08	1.74E+03
127184	Tetrachloroethylene	165.83	1.624	18.6	0.0184	8.20E-06	7.20E-02	6.976	1387	218	2.67	6.2	0.68	0	3.13E-08	2.00E+02
630206	1,1,1,2-Tetrachloroethane	167.85	1.59	12.03	0.00242	7.90E-06	7.10E-02	6.894	1355	192	2.63	6.2	0.68	0	5.81E-09	1.10E+03
924163	N-Nitrosodi-n-butylamine	158.24		0.03	0.00032	8.00E-06	8.00E-02			273	2.41	0.0001	1.00		1.00E-20	1.27E+03
930552	N-Nitrosopyrrolidine	100.12		0.092	1.2E-08	1.04E-05	7.36E-02			273	-0.19	0.0001	1.00		1.56E-08	1.00E+06
1319773	Cresols (total)	108.1	1.03	0.3	1.6E-06	9.30E-06	6.94E-02	8.850	2795	273	0	23	17.00	0	1.00E-20	2.20E+04
1330207	Xylenes	106.17	0.86	8.04178	0.00604	9.34E-06	7.14E-02	7.940	2090	273	3.17	40.8	1.80	0	2.43E-09	1.86E+02
1634044	Methyl tert-butyl ether	88	0.97	185.949	0.00056	1.05E-05	1.02E-01	6.852	1104	223	1.901	17.56	0.71	0	1.00E-20	3.88E+04
1746016	2,3,7,8-TCDD	322	1.41	7.4E-10	1.6E-05	8.00E-06	4.70E-02	6.977	2377	159	6.64	0.001	0.03	0	1.00E-20	1.90E-05
7439921	Lead	207.2		0	0 NA	NA				273	5.447				1.00E-20	
7439965	Manganese	54.938		0	0					273					1.00E-20	
7439976	Mercury	200.59		0.00196	0.0092	6.30E-06	3.07E-02			273	4.978				1.00E-20	5.62E-02
7440020	Nickel	58.69		0	0 NA	NA				273	1.914				1.00E-20	
7440382	Arsenic	74.92		0	0 NA	NA				273	1.462				1.00E-20	
7440393	Barium	137.33		0	0 NA	NA				273	2.724				1.00E-20	
7440417	Beryllium	9.012		0	0 NA	NA				273	1.845				1.00E-20	
7440439	Cadmium	112.41		0	0 NA	NA				273	2.21				1.00E-20	
7440473	Chromium (total)	51.996		0	0 NA	NA				273	1.255				1.00E-20	
7440484	Cobalt			0						273					1.00E-20	
7440622	Vanadium	50.94		0	0 NA	NA				273	1.699				1.00E-20	
10061015	cis-1,3-Dichloropropylene	110.97	1.2	32.8	0.00176	1.10E-05	5.85E-02	6.807	1328	230	2	10.76	0.76	0	9.81E-10	2.72E+03
10061026	trans-1,3-Dichloropropylene	110.97	1.2	23.3	0.00125	1.10E-05	5.85E-02	6.807	1328	230	2	10.76	0.76	0	9.81E-10	2.72E+03
16065831	Chromium (III)	51.996		0	0					273	1.255				1.00E-20	
18540299	Chromium (VI)	51.996		0	0	1.41E-05	2.00E-01			273	1.255				1.00E-20	

Appendix C

Sensitivity Analysis for Emissions Model



C. Sensitivity Analysis for Emissions Model

C.1 General Approach

A sensitivity analysis was performed on the CHEMDAT8 emission models used to estimate the emissions from the various waste management units (WMUs). As CHEMDAT8 employs exactly the same emission model equations for land application units, open landfills, and wastepiles, only a single sensitivity analysis was needed to encompass the variations for these units. However, three scenarios were modeled in the land application / open landfill / wastepile (or simply the LAU) emissions model sensitivity analysis to cover the range of assumptions that might be used in modeling these WMUs.

For a given WMU emission model, a base condition was defined and the annual emissions were estimated. Then, each of the emissions model input parameters were altered one at a time using a step function (either increasing or decreasing the input parameter by a factor of 2), and the annual emissions were compared with the base case annual emissions. For certain pairs of parameters, both input parameters were simultaneously altered to better illustrate the impact of indirect model parameters such as process residence time.

In addition to the emissions model input parameter, the emissions predictions are also expected to be a function of the contaminant's chemical and physical properties. Because constituent volatility is expected to have the greatest influence on the emissions estimates, the sensitivity analysis was performed for a number of constituents that range the spectrum of volatility.

C.2 Selection of Constituents for Sensitivity Analysis

Ten compounds were selected from the complete list of constituents to be modeled in the analysis based on a ranking of the constituent's Henry's law constant. The constituents were ranked using Henry's law constant because the most conservative volatile emissions estimates would result from aqueous wastes (rather than organic wastes, where constituent vapor pressure is the key parameter). The constituent properties database developed for the HWIR project was sorted and the constituents with the 5th, 15th, 25th, etc., to the 95th percentile Henry's law constants were selected for the analysis. The constituent properties database developed for the HWIR project was used as the primary source of the constituent-dependent input data. As the CHEMDAT8 model uses Monod kinetics for estimating the biodegradation rates in the tank model, the CHEMDAT8 model constituent database was used to supplement the HWIR constituent properties database (primarily for biodegradation rate constants, but also for hydrolysis and photolysis rates) prior to performing the sensitivity analysis. Table C-1 summarizes the constituent properties input data for the 10 constituents selected for the analysis. Except for the use of these 10 constituents in the sensitivity analysis, no other separate analysis was performed to assess the various models' sensitivity to a specific constituent input parameter.

There was one unusual compound in the selected group of constituents modeled. Phthalic anhydride reacts with water (undergoes hydrolysis) to form phthalic acid. Hydrolysis is only a loss mechanism in the surface impoundment and aerated tank models. The selection of a hydrolyzing compound was more by coincidence than by design, but it is interesting to note the impact (or the lack of impact) the model input parameters have for the predicted air emissions.

C.3 Sensitivity Analysis for Land Application Unit, Landfill, and Wastepile Emissions

Three potential modeling assumptions were investigated for the LAU emissions model sensitivity analysis as potential base cases. First, the LAU model was assumed to have biodegradation, and the waste (or soil waste mixture) was assumed to be an organic waste matrix. The base case LAU input parameters are provided in Table C-2. As seen in Table C-2, the LAU model has an input flag for biodegradation and aqueous waste matrix. For the second series of LAU emissions estimates, the biodegradation flag was set to zero (no biodegradation in an organic waste matrix). In the third series of LAU emissions estimates, the aqueous waste flag and the biodegradation flag were both set to 1 (biodegradation with an aqueous waste matrix). The results of the sensitivity model runs are summarized in Table C-3.

As stated previously, most of the parameters were altered by a factor of 2 in the step function analysis. For a few parameters, a factor of 2 change was not employed. Specifically, in reducing the air porosity, a reduced air porosity value of 0.2 was used (rather than $0.51 \div 2$). Additionally, the total porosity could only be lowered when the air porosity was lowered, and a value of 0.4 was used (rather than $0.6 \div 2$). Finally, an air temperature of 37 °F was used in the step function for the high-end air temperature (rather than 25×2). Note: For the air temperature step function increase, a value of 30 °F may have been more appropriate since the CHEMDAT8 model temperature adjustment uses a temperature of 20 °F as a reference temperature.

In reviewing the data in Table C-3, note that a value of -50 percent indicates a factor of 2 reduction in the annual emissions and a value of 100 percent indicates a factor of 2 increase in the annual emissions (i.e., direct proportionality provided a factor of 2 change in the input parameter) from the base case for that section of the table. The CHEMDAT8 LAU model is insensitive to windspeed for long-term emission estimates. Also, because the CHEMDAT8 model uses an application rate per volume of soil, simply increasing the surface area or the waste/till depth also increases the total quantity of waste applied. That is why the "low depth" emission runs do not show an increase in annual emissions (the total amount of contaminant applied is half that of the base case). Rerunning the "low depth" with a simultaneous increase in loading indicates a 50 percent increase in annual emissions for the organic waste matrix and the low volatile compounds, and base level of emissions for the volatile compounds in the aqueous waste matrix (i.e., nearly 100 percent volatilization).

In comparing the emissions results for the base case for the aqueous versus the organic waste matrix (Tables C-3a and C-3c), the emissions are an order of magnitude or several orders of magnitude higher for aqueous wastes than for organic wastes. Only acetone, which has a high volatility and is miscible in water, has comparable emission rates for the different waste matrices. The impact of including biodegradation can be seen by comparing the base emissions in

Tables C-3a and C-3b. The inclusion of biodegradation for the organic waste matrix reduced the annual emission rate (by a factor of approximately 4) for all compounds.

C.4 Sensitivity Analysis for Aerated Tank Emissions

Two basic modeling approaches were investigated for the aerated tank emission model sensitivity analysis: with biodegradation and without biodegradation. The base case aerated tank input parameters are provided in Table C-4. For the model runs without biodegradation, the active biomass input parameter was set to zero. Note: The CHEMDAT8 aerated tank emission model provides two different types of aeration (mechanical or submerged air) and a whole host of input parameters that affect aeration, especially mechanical aeration. Thus, a preliminary analysis was performed solely on the influence of altering the aeration input parameters when no biodegradation is present.

Table C-5 summarizes the sensitivity model runs for the aeration input parameters. For this analysis, mechanical aeration was primarily investigated. Submerged aeration has only one input parameter: submerged air flow rate. When submerged air flow was included, the fraction agitation was set to zero (no mechanical aeration). Due to the differences in the mass transfer mechanisms, some compounds may increase in emissions with submerged aeration (heptachlor epoxide at submerged air flow = $0.4 \text{ m}^3/\text{s}$), while others remain the same or have lower emissions.

For the aerated tank aeration parameter sensitivity analysis (Table C-5), the only input parameter that was not varied by a factor of 2 was the aerator power efficiency; an efficiency of 0.5 was used (rather than $0.83 \div 2$; a typical variation for this parameter is from 0.80 to 0.85). As seen in Table C-3, the aerated tank model is most sensitive to the fraction aerated, with the total power, power per aerator, and impeller diameter having some impact on the emission results. The other aeration input parameters (oxygen transfer rate, power efficiency, and impeller speed) had very little impact on the emission results.

Table C-6 summarizes the other input parameters for mechanically aerated tanks with and without active biomass. The only input parameter that was not varied by a factor of 2 was the influent solids content. The base case assumes no influent solids; the influent solids case assumed an influent solids concentration of 1 g/L. The emissions for the higher volatility compounds (ethyl ether and the dichloroethanes) increased by a factor of 2 when biodegradation was turned off; the emissions for the less volatile compounds, except for phthalic anhydride, increased by a factor of 4 to 100 when biodegradation was turned off. As phthalic anhydride is primarily removed by hydrolysis, the inclusion of biodegradation did not significantly alter its predicted emissions.

The impact of other model parameters can be seen by comparing the results within either Table C-6a or C-6b. However, it is interesting to see the difference in the impacts of some of the variables between the two table sections. For example, increasing the depth, which increases the residence time in the impoundment, reduces the emissions when biodegradation is present, but has no impact when biodegradation is not present (except for the hydrolyzing compound). On the other hand, increasing the surface area, which also increases the residence time, has little

impact on the emissions when biodegradation is present, but increases the emissions when biodegradation is absent for those compounds that do not already have emissions fractions over 80 or 90 percent. Note: At a fixed residence time, increasing the surface area (with an equal decrease in depth) increases the emissions regardless of the presence or absence of biodegradation. Increasing the flow rate also favors air emissions over biodegradation because it reduces the retention time at a given surface area.

Windspeed and influent solids have limited to no impact on the predicted emissions rates. Temperature, on the other hand, can have a very significant impact on the predicted emissions rates. Temperature impacts the air diffusivity raised to the 1.75 power. There is also a temperature-dependent correction factor used in CHEMDAT8 for the biodegradation rate. That is why the increased temperature reduced the air emissions for a few contaminants (those nearing 100 percent emissions) when biodegradation was present.

Table C-1. Constituent Input Parameters for the Emission Model Sensitivity Analysis

COMPOUND NAME	M.W.	DENSITY	VAP.PRESS	H LAW	DIFF.	DIFF. AIR	LOG	Kmax	K1	HYDROL.
		(g/cc)	(mmHg)	CONST	WATER	(cm2/sec)	OCT/ WATER PART. COEF			
ETHYL ETHER	74.12	0.71	7.07e-01	3.30e-02	9.30e-06	7.40e-02	0.83	17.56	0.57	0
DICHLOROETHANE(1,1)	98.96	1.17	2.99e-01	5.62e-03	1.05e-05	7.42e-02	1.79	10.76	2.3	0
DICHLOROETHANE 1,2	98.96	1.26	1.04e-01	9.79e-04	9.90e-06	1.04e-01	1.47	2.1	0.98	0
ACENAPHTHALENE	154.21	1.02	3.29e-06	1.55e-04	7.69e-06	4.21e-02	3.92	31.1	2.7	0
ACETONE	58.08	0.79	3.03e-01	3.88e-05	1.14e-05	1.24e-01	-0.24	1.3	1.15	0
HEPTACHLOR EPOXIDE	389.32	1.57	2.57e-08	9.50e-06	4.68e-06	1.22e-02	5.00	10.76	11.2	0
TOLUIDINE (o-)	107.16	0.989	4.21e-04	2.72e-06	9.12e-06	7.14e-02	1.34	31.1	0.86	0
DINITROPHENOL 2,4	184.11	1.68	6.71e-06	4.43e-07	9.06e-06	2.73e-02	1.55	8.0	0.62	0
PHTHALIC ANHYDRIDE	148.12	1.33	6.80e-07	1.63e-08	9.60e-06	7.10e-02	-0.62	17.56	0.078	5.55e-04
PHENYLENEDIAMINE (m-)	108.14	1.14	3.58e-06	1.35e-10	9.88e-06	6.63e-02	0.05	9.7	0.28	0

Table C-2. Land Application Model Input Parameters

INPUT PARAMETER	VALUE
L>Loading (g oil/cc soil)	0.036
Concentration in oil(ppmw)	2000
l,Depth of tilling (cm)	20
Total porosity	0.61
Air Porosity(0 if unknown)	0.5
MW oil	282
For aqueous waste, enter 1	0
Time of calc. (days)	365.25
For biodegradation,enter 1	1
Temperature (Deg. C)	25
Wind Speed (m/s)	4.47
Area (m2)	25000

Table C-3. Summary of Sensitivity Analysis for Land Application Model Emissions

C-3a. LAND APPLICATION // OPEN LANDFILL // WASTE PILE - WITH BIODEGRADATION - ORGANIC MATRIX													
COMPOUNDS	BASE	Percent Increase (or decrease) from BASE emissions											
	Annual Em.	lower	semi -	half	lower air	lower air & total	lower	higher	higher wind	low till depth	high till depth	low	low
	(Mg/yr)	loading	annual	conc.	porosity	porosity	MWt oil	temp	speed	high area	low area	area	depth
ETHYL ETHER	5.14e-02	-29%	41%	-50%	-86%	-72%	-29%	-4%	0%	100%	-50%	-50%	0%
DICHLOROETHANE(1,1)	4.27e-02	-29%	39%	-50%	-86%	-72%	-29%	-1%	0%	100%	-50%	-50%	0%
DICHLOROETHANE 1,2	5.21e-02	-27%	20%	-50%	-87%	-73%	-31%	22%	0%	87%	-51%	-50%	-6%
ACENAPHTHALENE	6.29e-05	-29%	41%	-50%	-86%	-72%	-29%	22%	0%	100%	-50%	-50%	0%
ACETONE	1.19e-01	-23%	41%	-50%	-89%	-78%	-39%	26%	0%	105%	-59%	-50%	2%
HEPTACHLOR EPOXIDE	5.08e-06	-29%	39%	-50%	-86%	-72%	-29%	35%	0%	100%	-50%	-50%	0%
TOLUIDINE (o-)	9.27e-04	-29%	41%	-50%	-86%	-72%	-29%	24%	0%	100%	-50%	-50%	0%
DINITROPHENOL 2,4	1.42e-04	-29%	36%	-50%	-86%	-72%	-29%	38%	0%	100%	-50%	-50%	0%
PHTHALIC ANHYDRIDE	4.94e-05	-29%	41%	-50%	-86%	-72%	-29%	19%	0%	100%	-50%	-50%	0%
PHENYLENEDIAMINE (m-)	1.47e-04	-29%	38%	-50%	-86%	-72%	-29%	19%	0%	100%	-50%	-50%	0%

C-3b. LAND APPLICATION // OPEN LANDFILL // WASTE PILE - WITHOUT BIODEGRADATION - ORGANIC MATRIX													
COMPOUNDS	BASE	Percent Increase (or decrease) from BASE emissions											
	Annual Em.	lower	semi -	half	lower air	lower air & total	lower	higher	higher wind	low till depth	high till depth	low	low
	(Mg/yr)	loading	annual	conc.	porosity	porosity	MWt oil	temp	speed	high area	low area	area	depth
ETHYL ETHER	1.95e-01	-32%	23%	-50%	-86%	-72%	-29%	24%	0%	69%	-50%	-50%	-15%
DICHLOROETHANE(1,1)	1.28e-01	-29%	32%	-50%	-86%	-72%	-29%	29%	0%	95%	-50%	-50%	-2%
DICHLOROETHANE 1,2	8.90e-02	-29%	38%	-50%	-86%	-72%	-29%	33%	0%	100%	-50%	-50%	0%
ACENAPHTHALENE	3.19e-04	-29%	50%	-50%	-86%	-72%	-29%	60%	0%	100%	-50%	-50%	0%
ACETONE	1.66e-01	-30%	27%	-50%	-86%	-72%	-29%	29%	0%	83%	-50%	-50%	-9%
HEPTACHLOR EPOXIDE	1.52e-05	-29%	50%	-50%	-86%	-72%	-29%	77%	0%	100%	-50%	-50%	0%
TOLUIDINE (o-)	4.70e-03	-29%	49%	-50%	-86%	-72%	-29%	63%	0%	100%	-50%	-50%	0%
DINITROPHENOL 2,4	3.67e-04	-29%	50%	-50%	-86%	-72%	-29%	80%	0%	100%	-50%	-50%	0%
PHTHALIC ANHYDRIDE	1.88e-04	-29%	50%	-50%	-86%	-72%	-29%	56%	0%	100%	-50%	-50%	0%
PHENYLENEDIAMINE (m-)	4.17e-04	-29%	50%	-50%	-86%	-72%	-29%	55%	0%	100%	-50%	-50%	0%

Table C-3. (Continued)

C-3c. LAND APPLICATION // OPEN LANDFILL // WASTE PILE - WITH BIODEGRADATION - AQUEOUS MATRIX													
	BASE	Percent Increase (or decrease) from BASE emissions											
COMPOUNDS	Annual Em.	lower	semi -	half	lower air	lower air & total	lower	higher	higher wind	low till depth	high till depth	low	low
	(Mg/yr)	loading	annual	conc.	porosity	porosity	MWt oil	temp	speed	high area	low area	area	depth
ETHYL ETHER	3.59e-01	-50%	0%	-50%	0%	0%	0%	0%	0%	0%	0%	-50%	-50%
DICHLOROETHANE(1,1)	3.59e-01	-50%	0%	-50%	0%	0%	0%	0%	0%	0%	0%	-50%	-50%
DICHLOROETHANE 1,2	3.59e-01	-50%	0%	-50%	0%	0%	0%	0%	0%	0%	0%	-50%	-50%
ACENAPHTHALENE	3.57e-01	-50%	0%	-50%	-21%	-10%	-1%	0%	0%	0%	-2%	-50%	-50%
ACETONE	3.59e-01	-50%	0%	-50%	-2%	-1%	0%	0%	0%	0%	0%	-50%	-50%
HEPTACHLOR EPOXIDE	3.26e-01	-48%	5%	-50%	-73%	-57%	-8%	4%	0%	7%	-20%	-50%	-46%
TOLUIDINE (o-)	3.06e-01	-46%	8%	-50%	-78%	-67%	-12%	5%	0%	12%	-28%	-50%	-44%
DINITROPHENOL 2,4	2.19e-01	-39%	22%	-50%	-85%	-77%	-22%	20%	0%	39%	-41%	-50%	-30%
PHTHALIC ANHYDRIDE	4.96e-02	-29%	41%	-50%	-86%	-79%	-29%	19%	0%	100%	-50%	-50%	0%
PHENYLENEDIAMINE (m-)	5.87e-03	-29%	38%	-50%	-86%	-79%	-29%	19%	0%	100%	-50%	-50%	0%

Table C-4. Aerated Tank Input Parameters for Base Case with Biodegradation

WINDSPEED	4.47	m/s			
DEPTH	4	m	Number impellers		
AREA	27	m2	Oxygen trans. rat.	3	lbO2/h-hp
FLOW	0.0075	m3/s	POWR (total)	7.5	HP
ACTIVE BIOMASS	2	g/l	Power efficiency	0.83	
BIOMASS SOLIDS IN	0	g/l	Temperature	25	deg C
VO INLET CONC.	10	mg/l	impeller dia	61	cm
TOTAL ORGANICS IN	250	mg/l	impeller speed	126	rad/s
TOTAL BIORATE	19	mg/g bio-hr			
FRACT. AGITATED	0.7				
SUBMERGED AIR FLOW	0	m3/s			

Table C-5. Aerated Tank Without Biodegradation- Sensitivity Analysis of Aeration Parameters

COMPOUNDS	BASE	Percent increase (or decrease) from BASE emissions								
	Annual Em.	2 Aerators	higher O2	lower power	lower fract	higher tot.	higher impeller	higher impeller	submerged air @ 0.2	submerged air @ 0.4
	(Mg/yr)		xfer rate	efficiency	agitated	power	diameter	speed	m3/sec	m3/sec
ETHYL ETHER	2.26e-01	0%	2%	-3%	-1%	2%	0%	0%	2%	3%
DICHLOROETHANE(1,1)	2.19e-01	-1%	2%	-2%	-4%	3%	-1%	-1%	-7%	0%
DICHLOROETHANE 1,2	1.89e-01	-5%	2%	-2%	-14%	6%	-5%	-2%	-35%	-15%
ACENAPHTHALENE	7.63e-02	-17%	1%	-1%	-38%	20%	-16%	-9%	-51%	-19%
ACETONE	4.12e-02	-20%	0%	0%	-41%	25%	-19%	-11%	-67%	-46%
HEPTACHLOR EPOXIDE	3.88e-03	-23%	0%	0%	-41%	30%	-21%	-12%	-21%	40%
TOLUIDINE (o-)	2.76e-03	-22%	0%	0%	-38%	29%	-21%	-12%	-51%	-26%
DINITROPHENOL 2,4	2.80e-04	-23%	0%	0%	-39%	30%	-21%	-12%	-37%	4%
PHTHALIC ANHYDRIDE	1.87e-06	-22%	0%	0%	-37%	29%	-21%	-12%	-49%	-24%
PHENYLENEDIAMINE (m-)	1.35e-07	-22%	0%	0%	-37%	29%	-21%	-12%	-48%	-22%

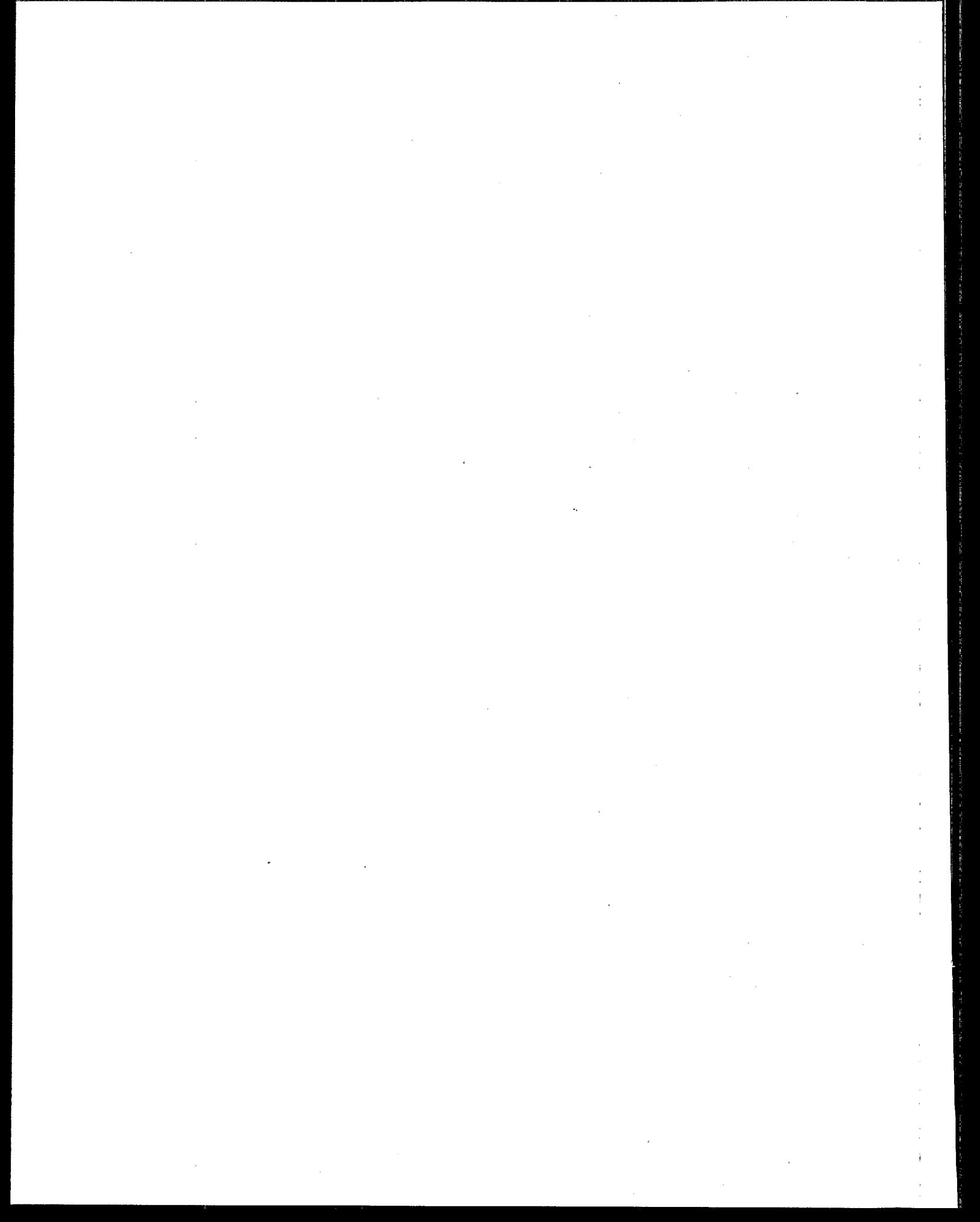
Table C-6. Summary of Sensitivity Analysis Model Runs for Aerated Tank Emissions Model

C-6a. AERATED TANK - WITH BIODEGRADATION

COMPOUNDS	BASE	Percent Increase (or decrease) from BASE emissions								
	Annual Emissions (Mg/yr)	higher biomass	higher tot. biorate	higher concentr.	larger depth	larger area	larger area half depth	influent solids	higher temp	higher flow rate
ETHYL ETHER	1.86e-01	-15%	0%	100%	-15%	-13%	2%	0%	-3%	93%
DICHLOROETHANE(1,1)	9.14e-02	-37%	0%	101%	-37%	-21%	19%	0%	-6%	95%
DICHLOROETHANE 1,2	7.54e-02	-38%	0%	104%	-38%	-7%	40%	-1%	2%	88%
ACENAPHTHALENE	4.88e-03	-48%	0%	101%	-48%	0%	92%	-5%	47%	92%
ACETONE	5.17e-03	-49%	0%	117%	-49%	1%	94%	-1%	-1%	92%
HEPTACHLOR EPOXIDE	4.38e-05	-50%	0%	102%	-50%	0%	99%	-6%	80%	100%
TOLUIDINE (o-)	3.55e-04	-47%	0%	101%	-47%	6%	99%	-1%	61%	78%
DINITROPHENOL 2,4	4.75e-05	-46%	0%	102%	-46%	8%	99%	-2%	101%	72%
PHTHALIC ANHYDRIDE	1.75e-06	-6%	0%	100%	-47%	5%	99%	-1%	132%	81%
PHENYLENEDIAMINE (m-)	4.15e-08	-41%	0%	101%	-41%	18%	99%	-3%	61%	53%

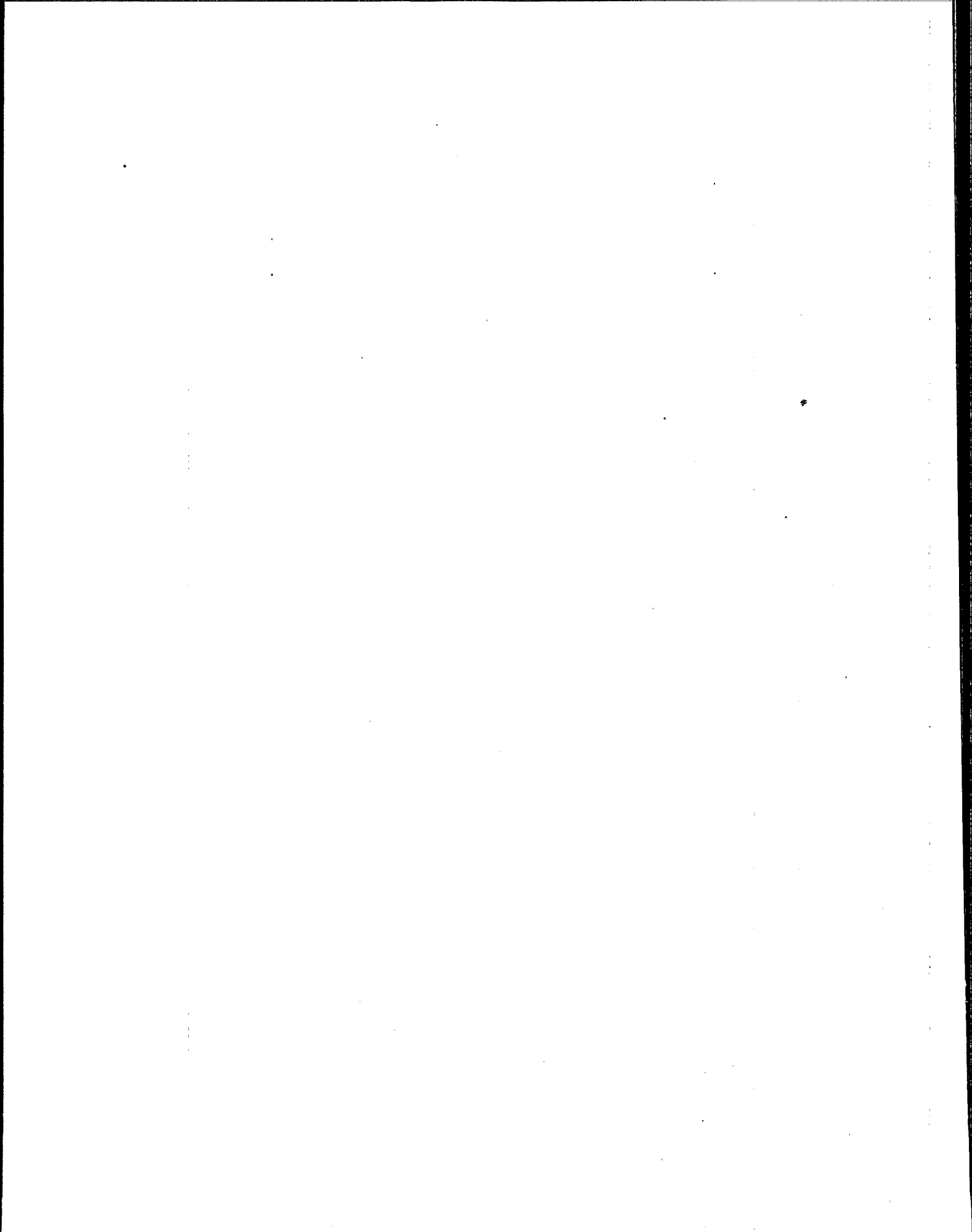
C-6b. AERATED TANK - WITHOUT BIODEGRADATION (no biomass)

COMPOUNDS	BASE	Percent Increase (or decrease) from BASE emissions								
	Annual Em. (Mg/yr)	higher concentr.	larger depth	larger area	larger area half depth	higher windspeed	influent solids	higher temp	higher flow rate	with biodegr.
ETHYL ETHER	2.26e-01	100%	0%	0%	0%	0%	-1%	1%	91%	-18%
DICHLOROETHANE(1,1)	2.19e-01	100%	0%	2%	2%	0%	-1%	3%	86%	-58%
DICHLOROETHANE 1,2	1.89e-01	100%	0%	9%	9%	0%	-2%	9%	67%	-60%
ACENAPHTHALENE	7.63e-02	100%	0%	50%	50%	2%	-44%	69%	19%	-94%
ACETONE	4.12e-02	100%	0%	69%	69%	5%	-8%	50%	10%	-87%
HEPTACHLOR EPOXIDE	3.88e-03	100%	0%	96%	96%	5%	-85%	198%	1%	-99%
TOLUIDINE (o-)	2.76e-03	100%	0%	97%	97%	6%	-10%	157%	1%	-87%
DINITROPHENOL 2,4	2.80e-04	100%	0%	99%	99%	5%	-11%	221%	0%	-83%
PHTHALIC ANHYDRIDE	1.87e-06	100%	-47%	6%	99%	6%	-1%	143%	80%	-7%
PHENYLENEDIAMINE (m-)	1.35e-07	100%	0%	99%	99%	6%	-9%	141%	0%	-69%



Appendix D

Sensitivity Analysis of ISC Air Model



D. Sensitivity Analysis of ISC Air Model

This appendix describes sensitivity analysis on depletion options, source shape and orientation, and receptor location and spacing.

D.1 Options With and Without Depletions

A sensitivity analysis was conducted using the ISCST3 model to determine whether dry and wet depletion options should be used in the risk analysis for five types of waste management units. A discussion of the analysis follows.

The depletion options (dry depletion and wet depletion) may be used with concentrations and depositions in the ISCST3 model runs. The model concentrations/depositions without depletion are higher than those with depletion. Because it takes much longer to run the ISCST3 model with depletions than without depletions, a sensitivity analysis was performed to investigate the differences of model outputs with and without selecting depletion options.

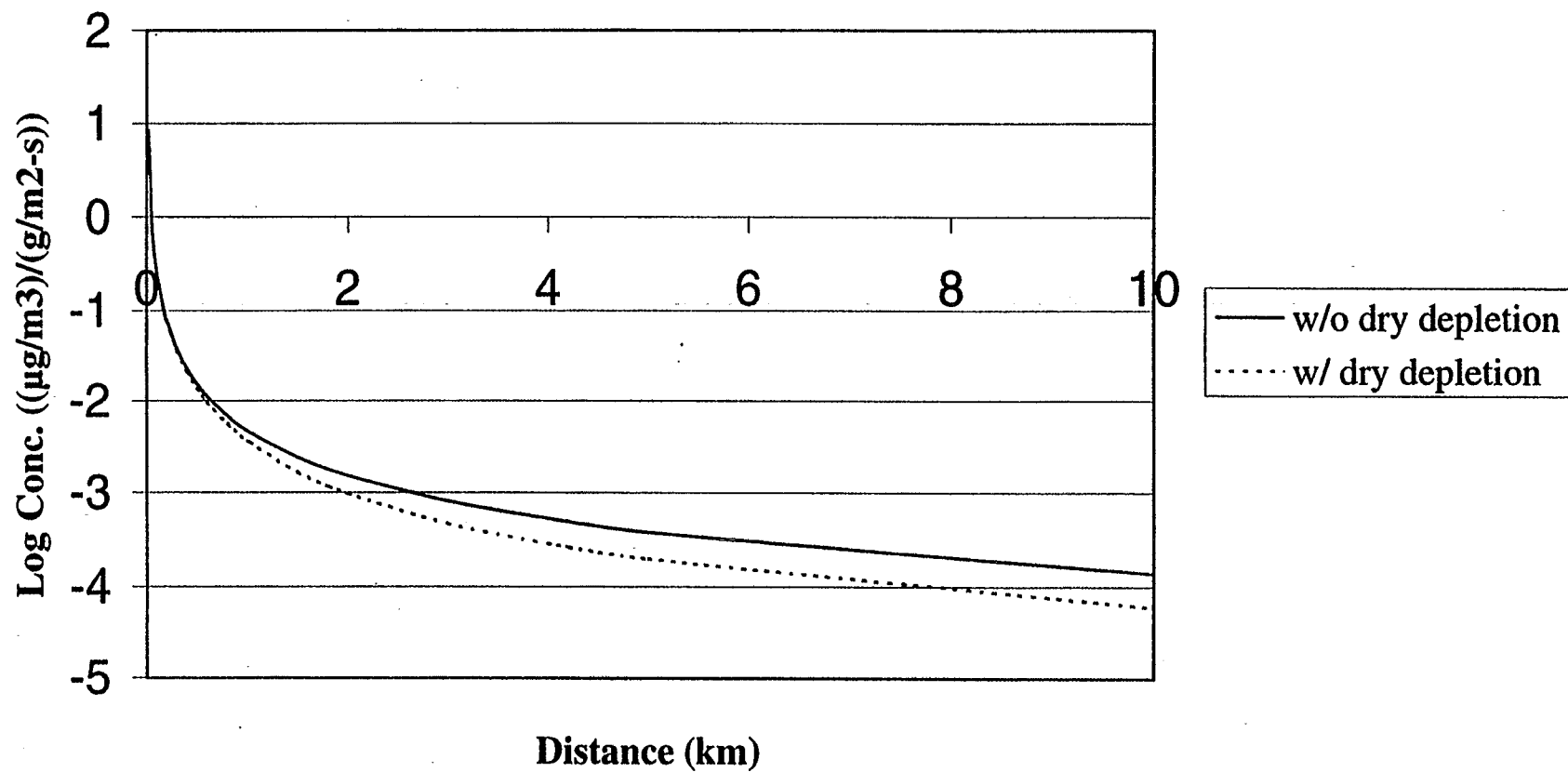
In this investigation, the 5th and the 95th percentile of sizes of LAUs were used to determine the relationship between concentrations with depletions and sizes of units.

For dry depletion, two meteorological stations (Little Rock, Arkansas, and Winnemucca, Nevada) were selected for the sensitivity analysis. The average particle sizes used in the sensitivity analysis are 20 μm and 5 μm with corresponding mass fraction of 50 percent each. The roughness length at application site was assumed as 0.4 meters.

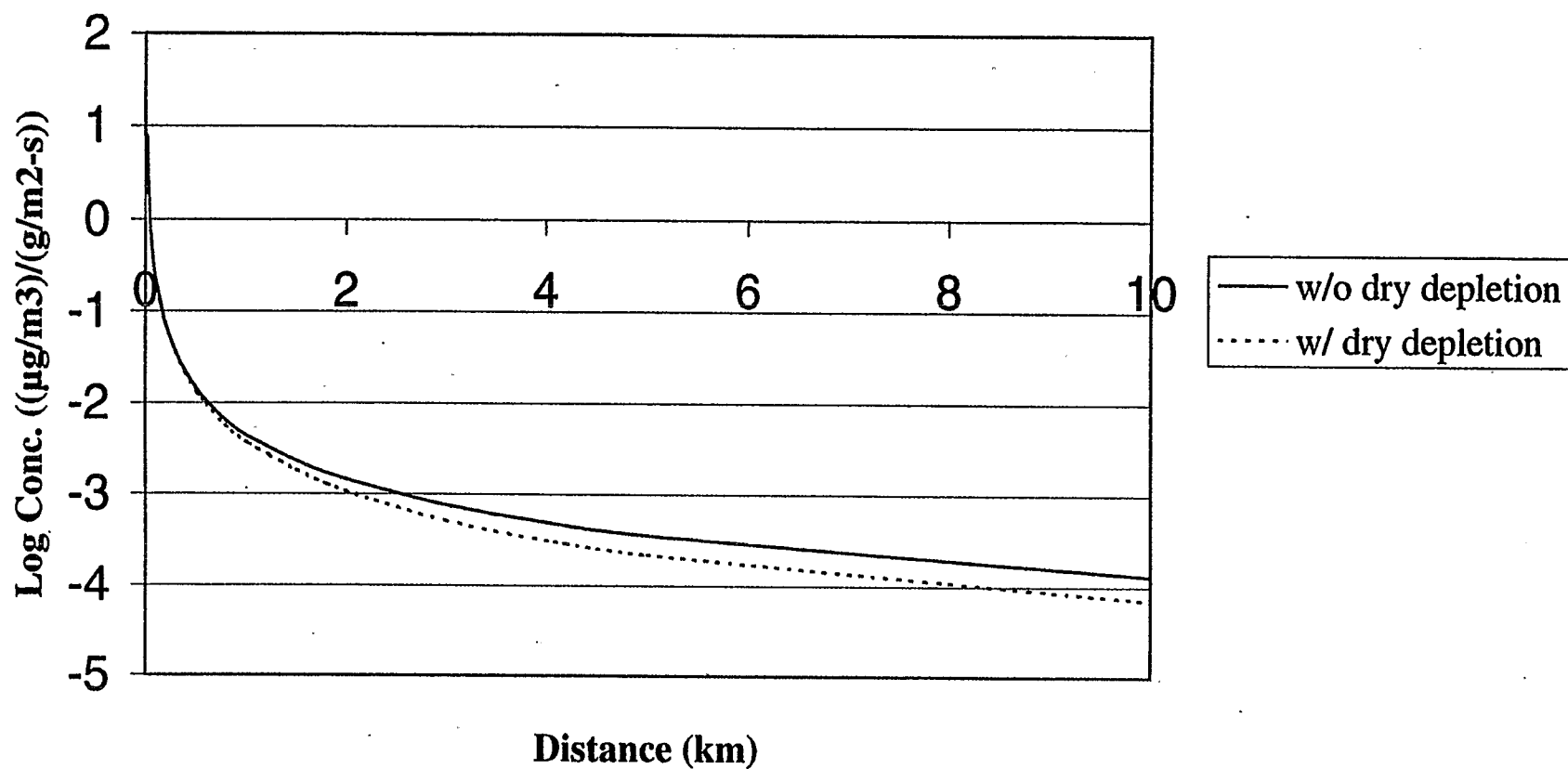
For wet depletion, two meteorological stations were selected for the sensitivity analysis: Atlanta, Georgia, with 49.8 inches precipitation per year (4th highest annual precipitation rate among the 29 meteorological stations to be modeled), and Winnemucca, Nevada, with 8.1 inches precipitation per year (3rd lowest annual precipitation rate). The reason for selecting a wet site and a dry site was to examine (1) whether wet depletion has a more significant impact for a wet site than a dry site; and (2) the differences of ambient concentrations that a very wet site can make with and without selecting wet depletion.

Five-year average concentrations with and without dry depletion were calculated using meteorological data from Little Rock and Winnemucca for the 5th and the 95th percentile of sizes of LAUs. The results show that the differences of the maximum concentrations with and without dry depletion are very small at close-to-source receptors. As the distance from the source increases, the differences between the dry depletion option and without dry depletion increase only slightly. The differences of concentrations are about 10 percent of the concentrations for the 95th percentile and are less than 2 percent of the concentrations for the 5th percentile at 50 meters from the edge of the LAU. The larger the area source, the larger the differences of the maximum concentrations. The results are shown in Figures D-1a through D-1d.

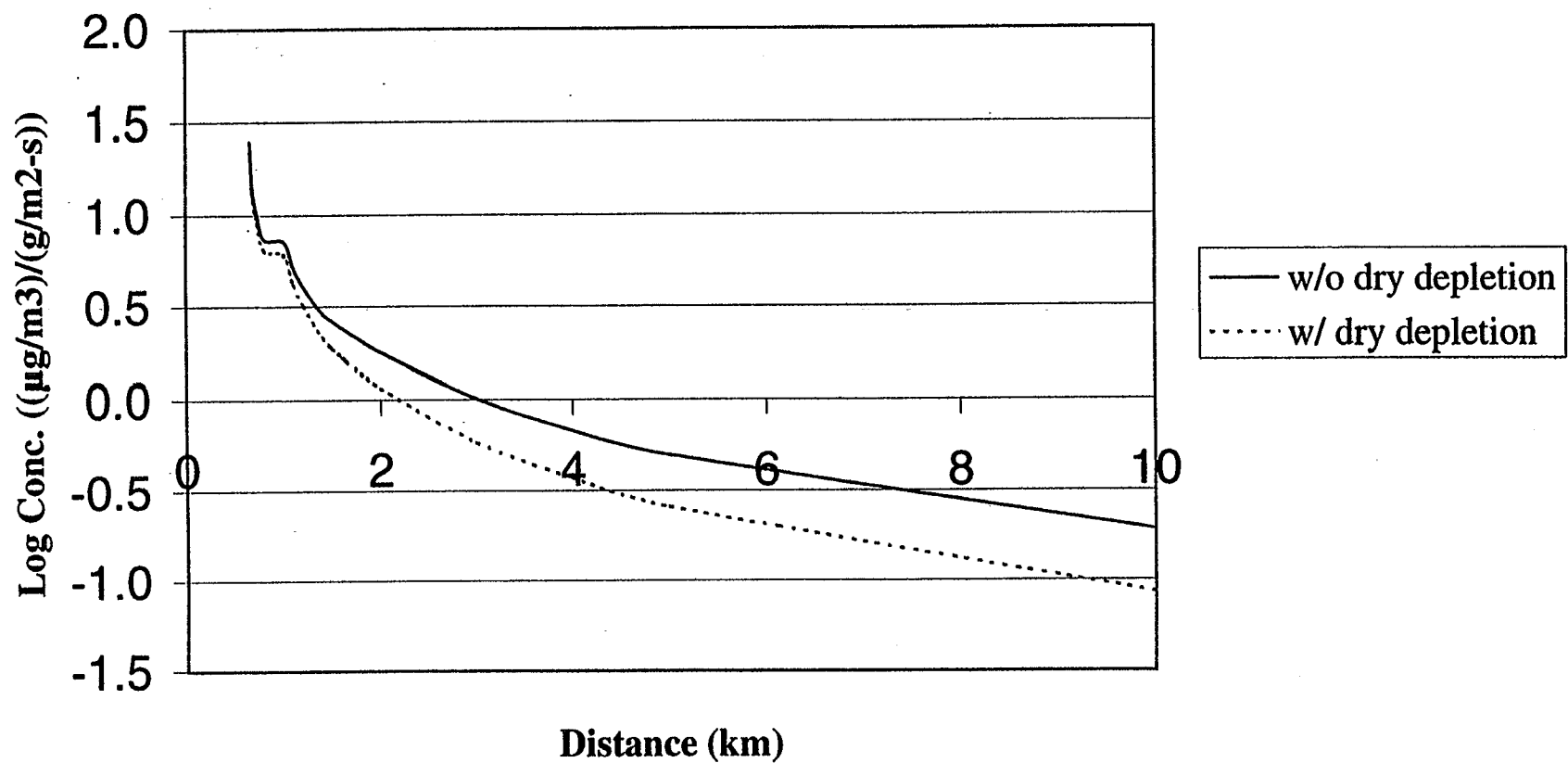
Five-year average concentrations with and without wet depletion also were calculated using meteorological data from Atlanta and Winnemucca for the 5th and 95th percentile of sizes of LAUs. The results show that the differences of the maximum concentrations with and without wet depletion are small for both Atlanta and Winnemucca sites. However, the differences in the maximum concentrations between the wet depletion option and without wet depletion are about 5 to 10 times greater for the Atlanta site than the Winnemucca site. Tables D-1a and D-1b show that for the 95th percentile unit size, at 50 meters from the edge of the unit, the differences in the maximum concentrations are only 0.03% and 0.37% for Winnemucca and Atlanta, respectively. This means that model concentrations with and without wet depletion are about the same.



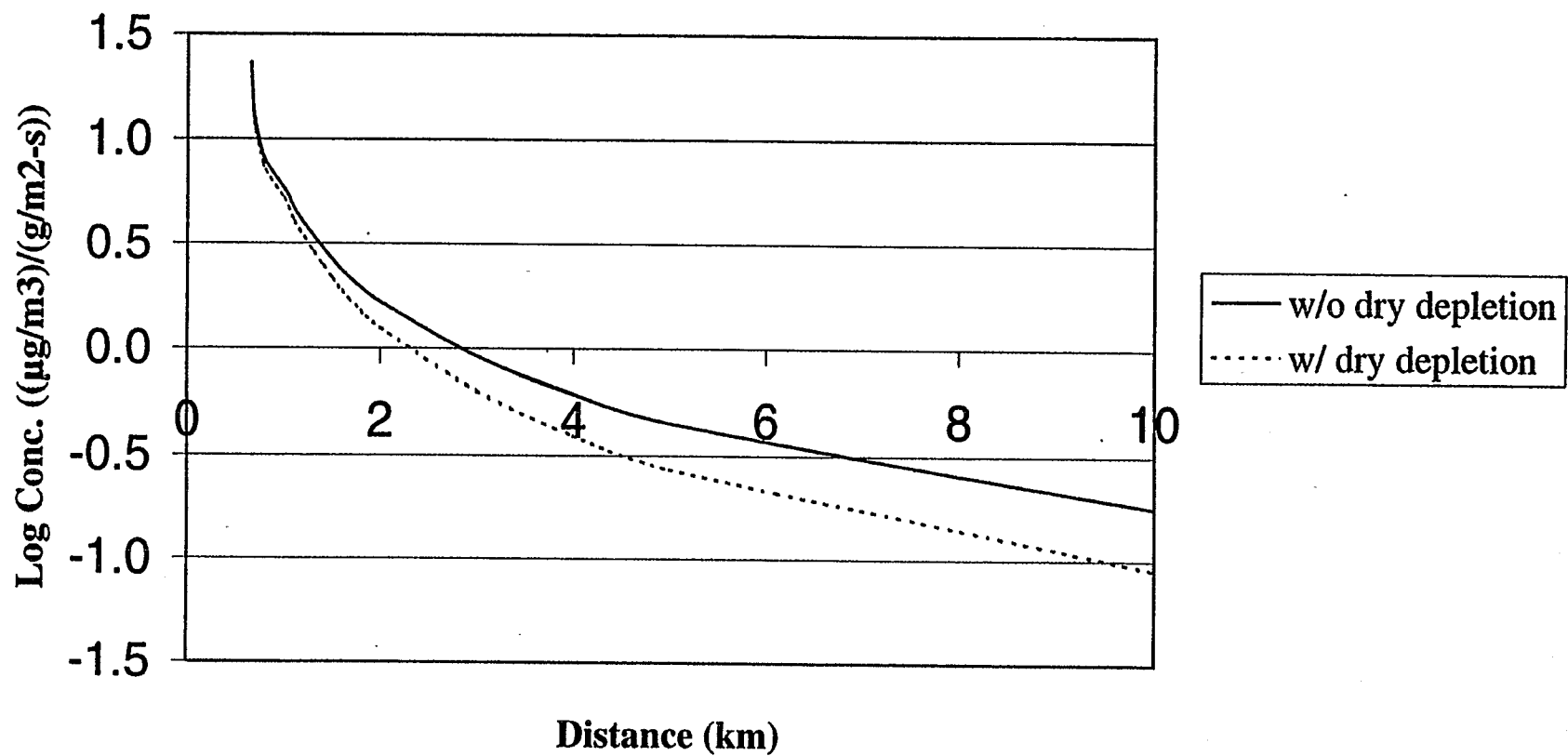
**Figure D-1a. Air Concentrations of Particles
(LAU, 5th Percentile, Little Rock, AR)**



**Figure D-lb. Air Concentrations of Particles
(LAU, 5th Percentile, Winnemucca, NV)**



**Figure D-1c. Air Concentrations of Particles
(LAU, 95th Percentile, Little Rock, AR)**



**Figure D-1d. Air Concentrations of Particles
(LAU, 95th Percentile, Winnemucca, NV).**

Table D-1a. Differences of Air Concentrations for Vapors Between Wet Depletion Option and Without Wet Depletion

(Atlanta, GA Site)

5th Percentile					95th Percentile				
Distance	w/o wet depletion Concentrations	w/ wet depletion Concentrations	Difference	Difference in	Distance	w/o wet depletion Concentrations	w/ wet depletion Concentrations	Difference	Difference in
(m)	(ug/m ³ / g/m ² -s)	(ug/m ³ / g/m ² -s)	(ug/m ³ / g/m ² -s)	Percentage	(m)	(ug/m ³ / g/m ² -s)	(ug/m ³ / g/m ² -s)	(ug/m ³ / g/m ² -s)	Percentage
19.3 ⁽¹⁾	7.40752	7.40716	0.00036	0.005%	651.9 ⁽¹⁾	0.00614	0.00612	0.00002	0.33%
47.3 ⁽¹⁾	0.93175	0.93159	0.00016	0.017%	676.9 ⁽¹⁾	0.00574	0.00573	0.00001	0.17%
75.2 ⁽¹⁾	0.38178	0.38168	0.00010	0.026%	701.9 ⁽¹⁾	0.00539	0.00537	0.00002	0.37%
100	0.25129	0.25121	0.00008	0.032%	726.9 ⁽¹⁾	0.00507	0.00505	0.00002	0.39%
103.2 ⁽¹⁾	0.21003	0.20996	0.00007	0.033%	801.9 ⁽¹⁾	0.00427	0.00426	0.00001	0.23%
187.0 ⁽¹⁾	0.06886	0.06882	0.00004	0.058%	1000	0.00400	0.00399	0.00001	0.25%
200	0.07091	0.07086	0.00005	0.071%	1100	0.00342	0.00341	0.00001	0.29%
300	0.03390	0.03387	0.00003	0.088%	1200	0.00296	0.00295	0.00001	0.34%
400	0.02026	0.02024	0.00002	0.099%	1300	0.00260	0.00259	0.00001	0.38%
500	0.01359	0.01357	0.00002	0.147%	1400	0.00230	0.00229	0.00001	0.43%
600	0.00981	0.00979	0.00002	0.204%	1500	0.00205	0.00205	0.00000	0.00%
800	0.00590	0.00589	0.00001	0.169%	1600	0.00185	0.00184	0.00001	0.54%
1000	0.00400	0.00399	0.00001	0.250%	1800	0.00152	0.00152	0.00000	0.00%
1500	0.00205	0.00205	0.00000	0.000%	2000	0.00128	0.00128	0.00000	0.00%
2000	0.00128	0.00128	0.00000	0.000%	3000	0.00068	0.00067	0.00001	1.47%
3000	0.00068	0.00067	0.00001	1.471%	4000	0.00044	0.00043	0.00001	2.27%
4000	0.00044	0.00043	0.00001	2.273%	5000	0.00031	0.00031	0.00000	0.00%
5000	0.00031	0.00031	0.00000	0.000%	10000	0.00011	0.00011	0.00000	0.00%
10000	0.00011	0.00011	0.00000	0.000%					

⁽¹⁾ These refer to the distances from the center of emission source to the maximum concentration points along 0, 25, 50, 75, and 150 meter receptor squares, respectively.

Table D-1b. Differences of Air Concentrations for Vapors Between Wet Depletion Option and Without Wet Depletion

(Winnemucca, NV Site)

5th Percentile					95th Percentile				
Distance (m)	w/o wet depletion Concentrations (ug/m ³ / g/m ² -s)	w/ wet depletion Concentrations (ug/m ³ / g/m ² -s)	Difference (ug/m ³ / g/m ² -s)	Difference in Percentage	Distance (m)	w/o wet depletion Concentrations (ug/m ³ / g/m ² -s)	w/ wet depletion Concentrations (ug/m ³ / g/m ² -s)	Difference (ug/m ³ / g/m ² -s)	Difference in Percentage
17.3 ⁽¹⁾	7.79132	7.79125	0.00007	0.001%	651.9 ⁽¹⁾	23.14326	23.13885	0.00441	0.02%
42.3 ⁽¹⁾	1.08468	1.08464	0.00004	0.004%	676.9 ⁽¹⁾	13.86979	13.86551	0.00428	0.03%
67.3 ⁽¹⁾	0.48369	0.48367	0.00002	0.004%	701.9 ⁽¹⁾	11.62889	11.62486	0.00403	0.03%
92.3 ⁽¹⁾	0.27965	0.27963	0.00002	0.007%	726.9 ⁽¹⁾	10.25373	10.24985	0.00388	0.04%
100	0.24315	0.24313	0.00002	0.008%	801.9 ⁽¹⁾	7.84900	7.84548	0.00352	0.04%
167.3 ⁽¹⁾	0.09949	0.09948	0.00001	0.010%	1000	5.85241	5.84988	0.00253	0.04%
200	0.07296	0.07295	0.00001	0.014%	1100	4.69239	4.68991	0.00248	0.05%
300	0.03600	0.03599	0.00001	0.028%	1200	3.98357	3.98130	0.00227	0.06%
400	0.02181	0.02180	0.00001	0.046%	1300	3.43255	3.43045	0.00210	0.06%
500	0.01475	0.01474	0.00001	0.068%	1400	2.99083	2.98887	0.00196	0.07%
600	0.01070	0.01070	0.00000	0.000%	1500	2.63019	2.62837	0.00182	0.07%
800	0.00649	0.00648	0.00001	0.154%	1600	2.33211	2.33042	0.00169	0.07%
1000	0.00443	0.00443	0.00000	0.000%	1800	1.93762	1.93554	0.00208	0.11%
1500	0.00229	0.00229	0.00000	0.000%	2000	1.65686	1.65487	0.00199	0.12%
2000	0.00144	0.00144	0.00000	0.000%	3000	0.91889	0.91727	0.00162	0.18%
3000	0.00077	0.00077	0.00000	0.000%	4000	0.61160	0.61020	0.00140	0.23%
4000	0.00050	0.00050	0.00000	0.000%	5000	0.45013	0.44890	0.00123	0.27%
5000	0.00036	0.00036	0.00000	0.000%	10000	0.17843	0.17767	0.00076	0.43%
10000	0.00013	0.00013	0.00000	0.000%					

⁽¹⁾ These refer to the distances from the center of emission source to the maximum concentration points along 0, 25, 50, 75, and 150 meter receptor squares, respectively.

D.2 Source Shape and Orientation

A sensitivity analysis was conducted using the ISCST3 air model to determine what role source shape and orientation play in determining dispersion coefficients of air pollutants. A discussion of this analysis follows.

Three different sources were chosen for this analysis. The sources were a square (source No. 1), a rectangle oriented east to west (source No. 2), and a rectangle oriented north to south (source No. 3). All three sources had an area of 400 m² in order to ensure that equal emission rates were compared. The rectangles were selected to be exactly two times longer and half as wide as the square (see Figure D-2).

Two meteorological stations at Little Rock, Arkansas, and Los Angeles, California, were selected for this modeling analysis in order to compare two different meteorological regimes. Little Rock was selected because of its evenly distributed wind directions and Los Angeles was selected because it has a predominantly southwest wind direction (see Figure D-3). Five years of meteorological data were used for this analysis.

Each area source was modeled with similar receptor grids to ensure consistency. Sixteen receptors were placed on the edge of each of the area sources and another 16 were placed 25 meters out from the edge. Each of these two receptor groups were modeled as a Cartesian receptor grid. Two receptor rings were also placed at 50 and 100 meters out from the center of the source. This polar receptor grid consisted of 16 receptors with a 22.5 degree interval between receptors. See Figures D-4a through D-4c for receptor locations.

The ISCST3 model was run using the meteorological data from Little Rock, Arkansas, and Los Angeles, California, and the results are shown in Tables D-2a and D-2b. The results indicated that the standard deviation of the differences in air concentrations is greatest between source No. 2 and source No. 3. This difference is due to the orientation of the source. This occurs for both the Cartesian receptor grid and the polar receptor grid at both meteorological locations. This shows that the model is sensitive to the orientation of the rectangular area source.

Standard deviations are significantly smaller when source No. 1 is compared to source Nos. 2 or 3. This shows that the differences in Unitized Air Concentration (UAC) between the square source and the two rectangular sources are less than the differences between the two rectangular sources. A square area source also contributes the least amount of impact of orientation. Since no information on source shape or orientation is available, a square source will minimize the errors caused by different source shapes and orientations.

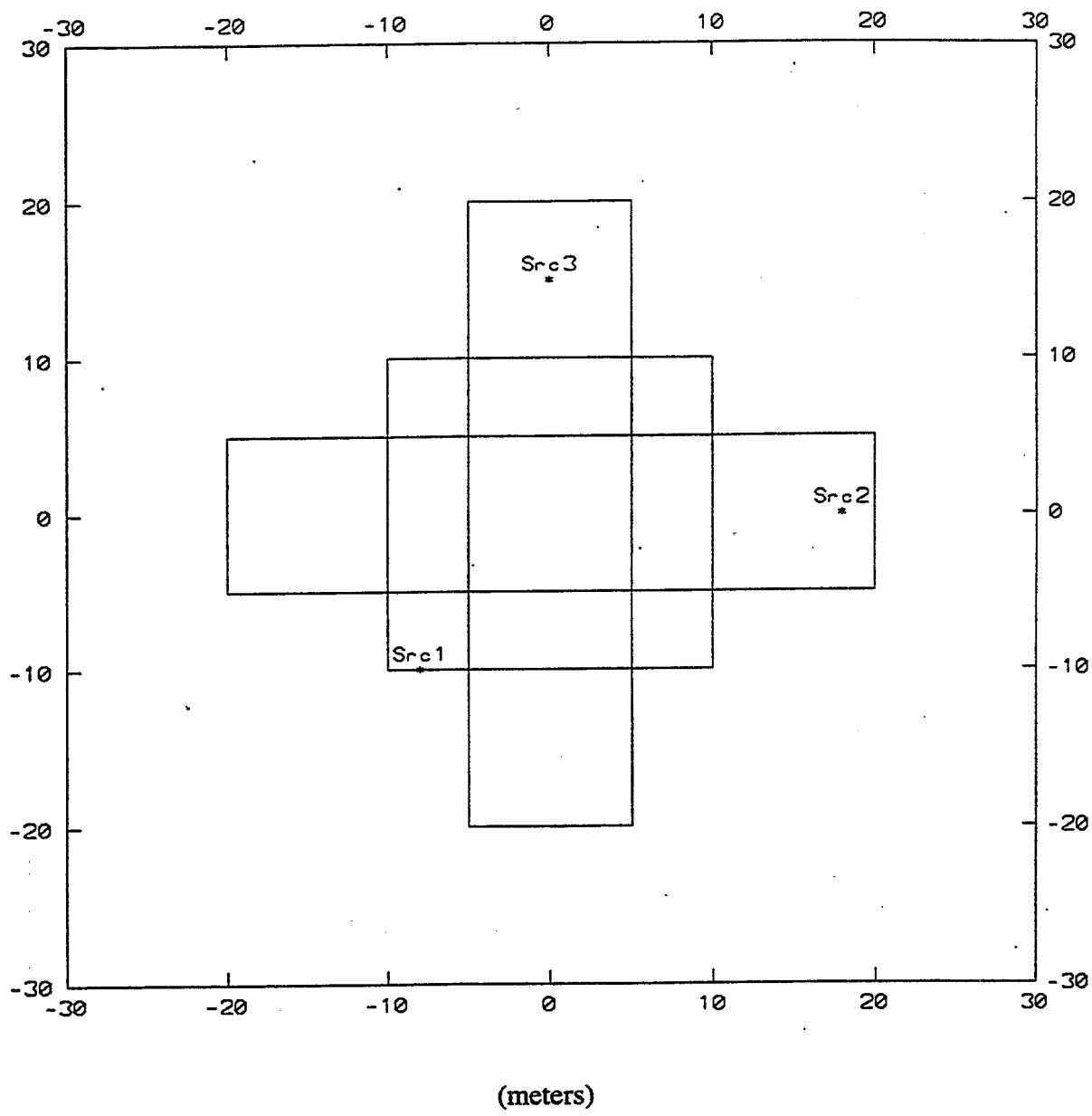
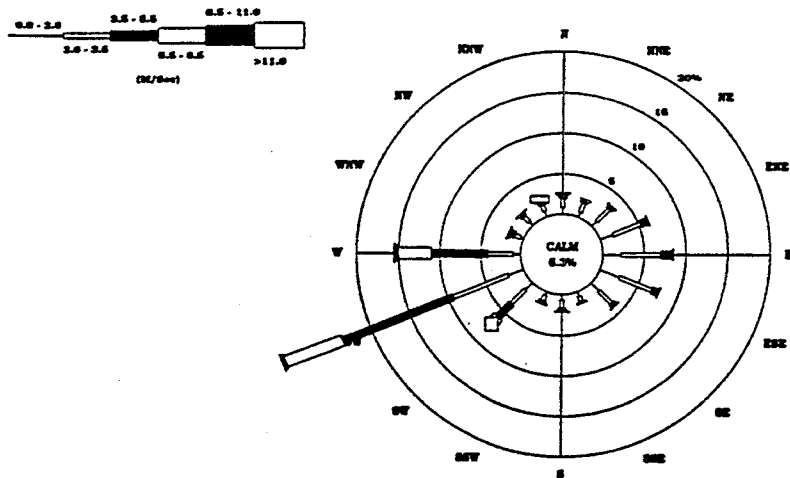


Figure D-2. Source Shapes and Orientations

Los Angeles, California



Little Rock, Arkansas

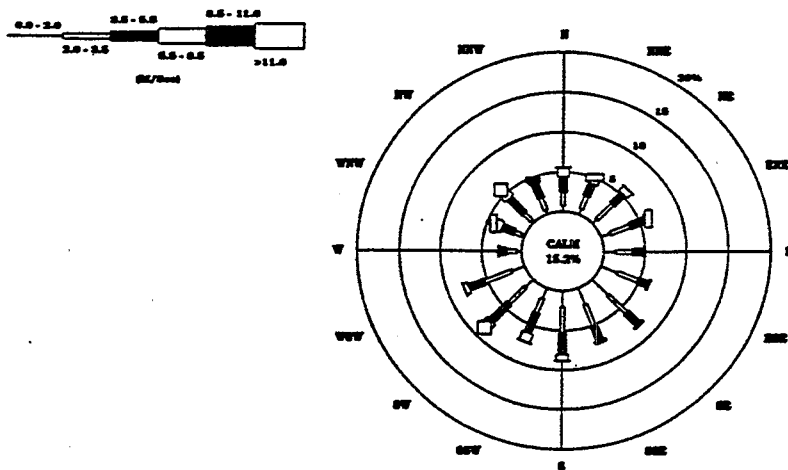


Figure D-3. Wind Roses

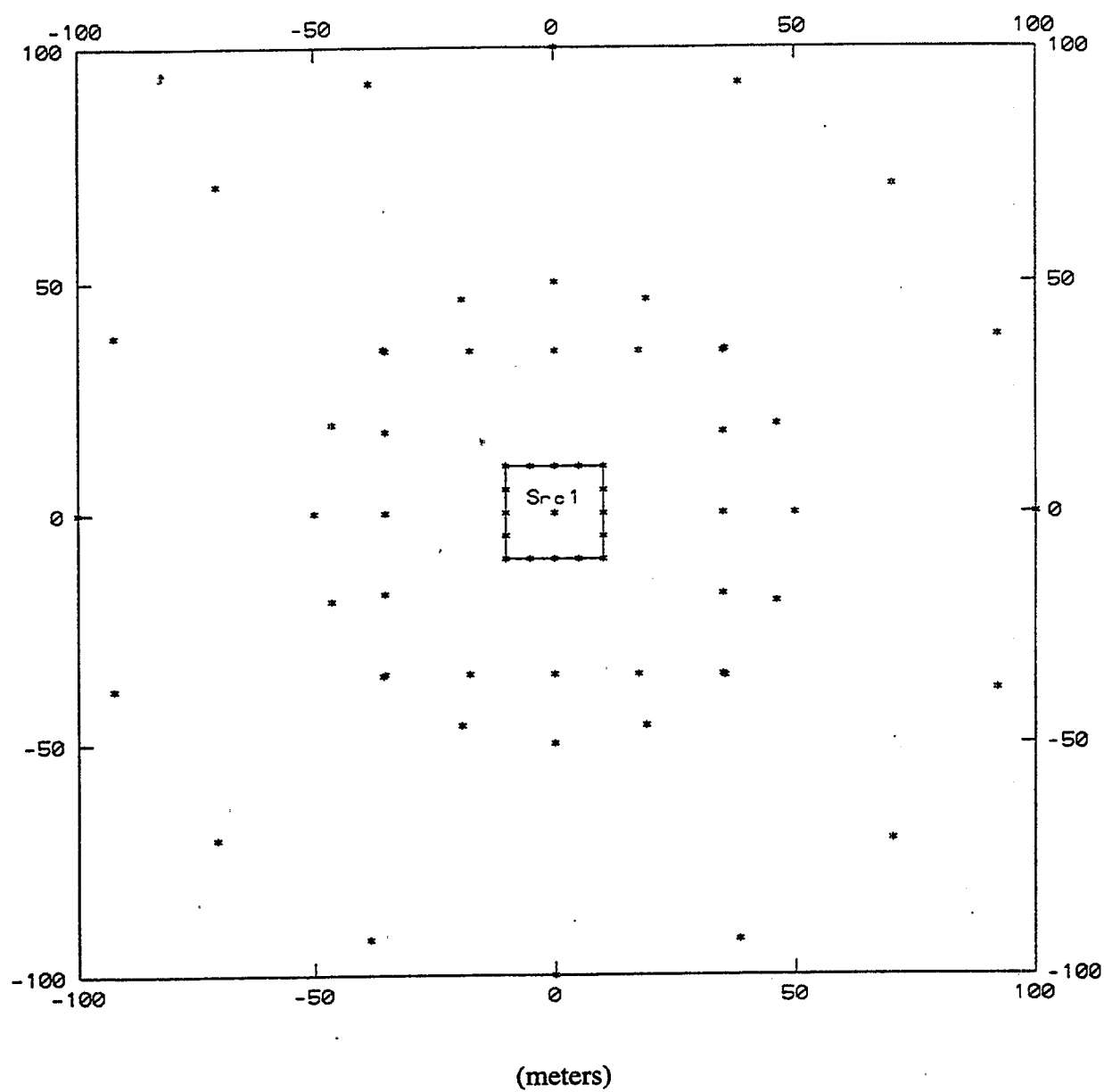


Figure D-4a. Receptor Locations (Source No. 1)

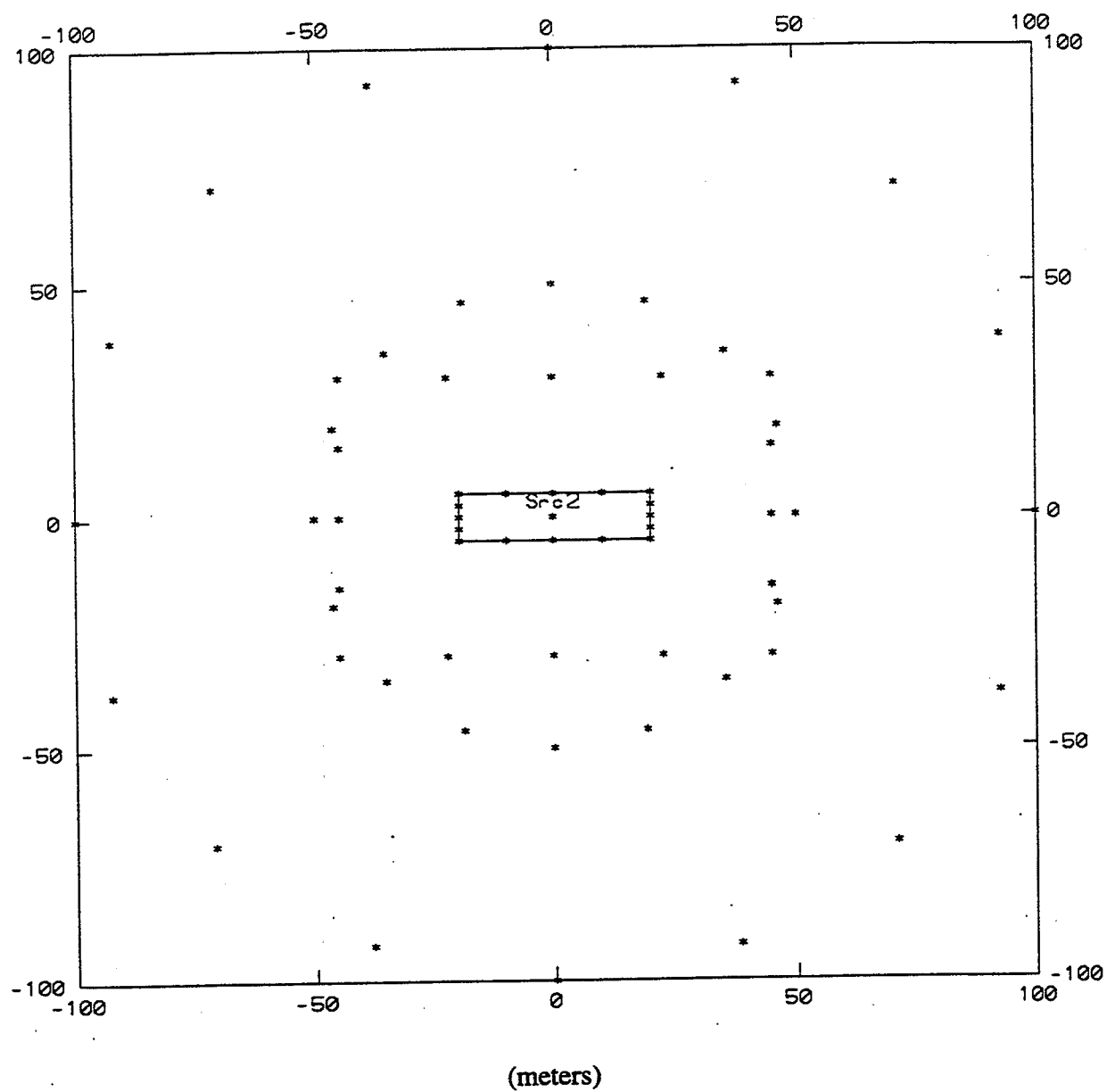


Figure D-4b. Receptor Locations (Source No. 2)

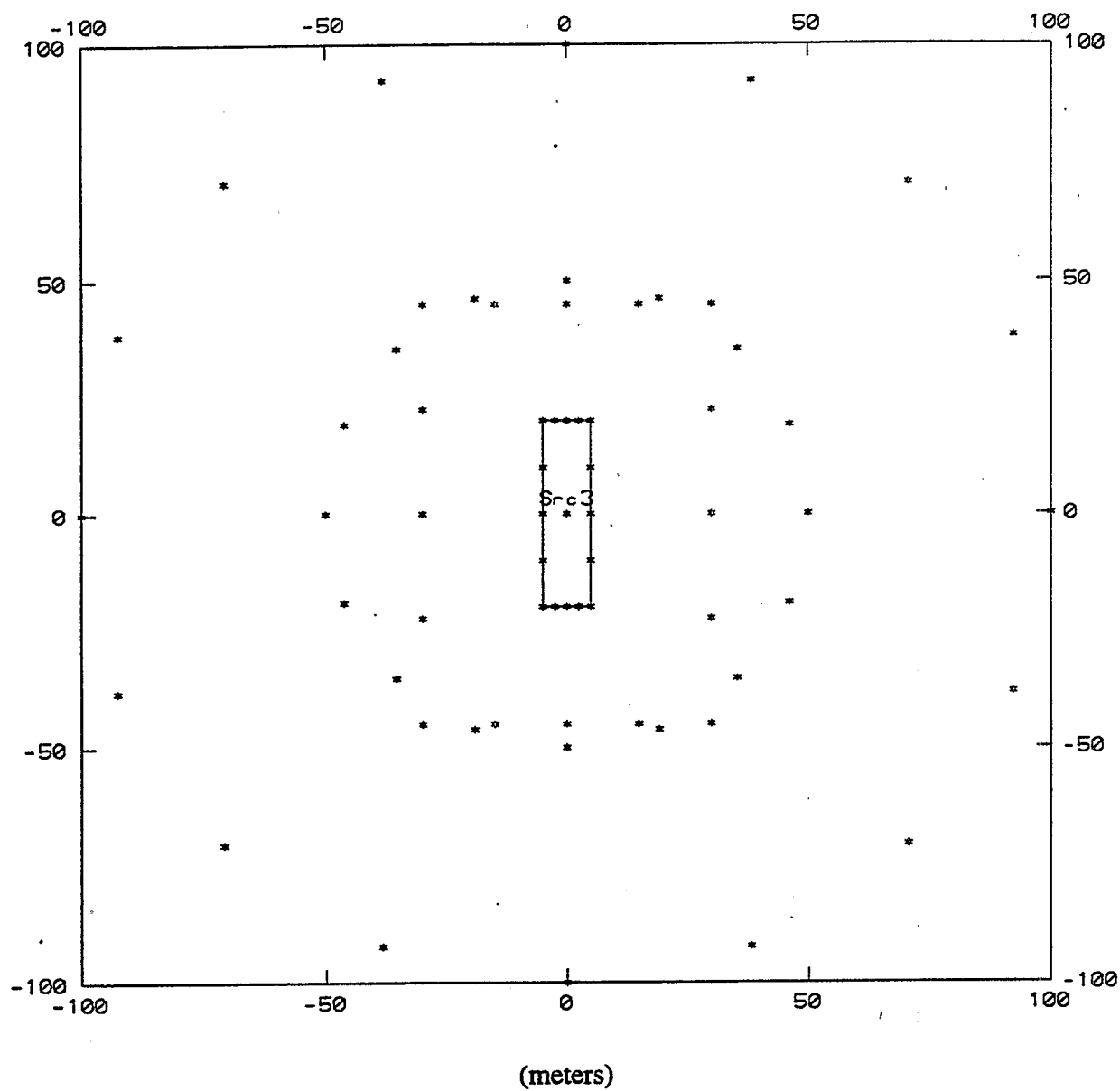


Figure D-4c. Receptor Locations (Source No. 3)

Table D-2a. Comparisons of Unitized Air Concentrations ($\mu\text{g}/\text{m}^3$ / $\mu\text{g}/\text{s}\cdot\text{m}^2$) for Different Source Shapes and Orientations
(Little Rock, Arkansas)

Source No. 1 (20m x 20m)			Source No. 2 (40m x 10m)			Source No. 3 (10m x 40m)			Differences in UACs Sources No. 1 and No. 2		Differences in UACs Sources No. 1 and No. 3		Differences in UACs Sources No. 2 and No. 3	
Polar Receptor Grid									Diff. In UAC % of Diff.		Diff. In UAC % of Diff.		Diff. In UAC % of Diff.	
X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	X (m)	Y (m)	UAC						
19	46	0.190	19	46	0.199	19	46	0.211	0.010	5%	0.021	11%	0.012	6%
38	92	0.050	38	92	0.051	38	92	0.051	0.001	1%	0.001	2%	0.000	1%
35	35	0.249	35	35	0.243	35	35	0.278	-0.007	-3%	0.028	11%	0.035	14%
71	71	0.067	71	71	0.067	71	71	0.069	-0.001	-1%	0.001	2%	0.002	3%
46	19	0.321	46	19	0.361	46	19	0.256	0.041	13%	-0.065	-20%	-0.105	-29%
92	38	0.095	92	38	0.098	92	38	0.088	0.003	3%	-0.007	-7%	-0.010	-10%
50	0	0.124	50	0	0.128	50	0	0.147	0.004	3%	0.023	19%	0.020	15%
100	0	0.030	100	0	0.030	100	0	0.033	0.000	-1%	0.003	9%	0.003	11%
46	-19	0.085	46	-19	0.096	46	-19	0.084	0.011	12%	-0.001	-1%	-0.011	-12%
92	-38	0.023	92	-38	0.024	92	-38	0.023	0.001	2%	-0.001	-2%	-0.001	-5%
35	-35	0.106	35	-35	0.109	35	-35	0.103	0.003	3%	-0.003	-3%	-0.006	-6%
71	-71	0.030	71	-71	0.030	71	-71	0.029	0.000	0%	0.000	-1%	-0.001	-2%
19	-46	0.117	19	-46	0.113	19	-46	0.128	-0.005	-4%	0.011	9%	0.016	14%
38	-92	0.033	38	-92	0.032	38	-92	0.034	-0.001	-4%	0.001	2%	0.002	7%
0	-50	0.122	0	-50	0.117	0	-50	0.143	-0.005	-4%	0.021	17%	0.026	22%
0	-100	0.035	0	-100	0.033	0	-100	0.037	-0.002	-5%	0.002	5%	0.004	11%
-19	-46	0.134	-19	-46	0.128	-19	-46	0.150	-0.006	-4%	0.016	12%	0.022	17%
-38	-92	0.038	-38	-92	0.036	-38	-92	0.038	-0.002	-4%	0.001	2%	0.002	6%
-35	-35	0.161	-35	-35	0.158	-35	-35	0.170	-0.003	-2%	0.009	6%	0.012	8%
-71	-71	0.043	-71	-71	0.043	-71	-71	0.045	0.000	1%	0.001	3%	0.001	3%
-46	-19	0.159	-46	-19	0.185	-46	-19	0.140	0.026	16%	-0.019	-12%	-0.045	-24%
-92	-38	0.044	-92	-38	0.046	-92	-38	0.043	0.002	4%	-0.002	-4%	-0.004	-8%
-50	0	0.103	-50	0	0.114	-50	0	0.107	0.011	11%	0.004	4%	-0.007	-6%
-100	0	0.027	-100	0	0.027	-100	0	0.027	0.000	2%	0.000	1%	0.000	0%
-46	19	0.126	-46	19	0.145	-46	19	0.118	0.019	15%	-0.008	-6%	-0.027	-18%
-92	38	0.035	-92	38	0.036	-92	38	0.034	0.001	4%	-0.001	-4%	-0.003	-7%
-35	35	0.152	-35	35	0.160	-35	35	0.153	0.008	5%	0.001	0%	-0.007	-5%
-71	71	0.041	-71	71	0.042	-71	71	0.041	0.001	3%	0.001	2%	-0.001	-2%
-19	46	0.173	-19	46	0.179	-19	46	0.187	0.007	4%	0.014	8%	0.008	4%
-38	92	0.047	-38	92	0.047	-38	92	0.048	0.000	0%	0.001	3%	0.001	3%
0	50	0.224	0	50	0.191	0	50	0.276	-0.032	-14%	0.052	23%	0.085	44%
0	100	0.068	0	100	0.061	0	100	0.074	-0.008	-11%	0.006	9%	0.014	22%
Standard Deviation:									0.012	7%	0.018	9%	0.028	14%

Table D-2a. Comparisons of Unitized Air Concentrations ($\mu\text{g}/\text{m}^3$ / $\mu\text{g}/\text{s}\cdot\text{m}^2$) for Different Source Shapes and Orientations
(Little Rock, Arkansas)

Source No. 1 (20m x 20m)			Source No. 2 (40m x 10m)			Source No. 3 (10m x 40m)			Differences in UACs Sources No. 1 and No. 2		Differences in UACs Sources No. 1 and No. 3		Differences in UACs Sources No. 2 and No. 3	
Cartesian Receptor Grid									Diff. In UAC		Diff. In UAC		Diff. In UAC	
X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	% of Diff.	% of Diff.	% of Diff.	% of Diff.	% of Diff.	
-10	-10	3.014	-20	-5	2.675	-5	-20	2.673	-0.339	-11%	-0.341	-11%	-0.002	0%
-5	-10	4.266	-10	-5	4.219	-2.5	-20	3.451	-0.047	-1%	-0.815	-19%	-0.769	-18%
0	-10	4.354	0	-5	4.307	0	-20	3.526	-0.047	-1%	-0.827	-19%	-0.781	-18%
5	-10	3.961	10	-5	4.069	2.5	-20	3.152	0.109	3%	-0.809	-20%	-0.918	-23%
10	-10	2.175	20	-5	1.899	5	-20	2.011	-0.276	-13%	-0.164	-8%	0.112	6%
10	-5	5.211	20	-2.5	3.875	5	-10	5.567	-1.337	-26%	0.355	7%	1.692	44%
10	0	5.968	20	0	4.704	5	0	5.913	-1.264	-21%	-0.055	-1%	1.209	26%
10	5	6.012	20	2.5	4.918	5	10	5.834	-1.094	-18%	-0.178	-3%	0.916	19%
10	10	4.946	20	5	4.468	5	20	4.344	-0.477	-10%	-0.602	-12%	-0.125	-3%
5	10	6.804	10	5	6.758	2.5	20	5.550	-0.047	-1%	-1.254	-18%	-1.208	-18%
0	10	6.846	0	5	6.830	0	20	5.604	-0.016	0%	-1.242	-18%	-1.226	-18%
-5	10	6.157	-10	5	6.353	-2.5	20	4.954	0.196	3%	-1.203	-20%	-1.399	-22%
-10	10	3.245	-20	5	2.793	-5	20	3.052	-0.451	-14%	-0.193	-6%	0.259	9%
-10	5	4.923	-20	2.5	3.801	-5	10	5.166	-1.121	-23%	0.244	5%	1.365	36%
-10	0	5.169	-20	0	4.032	-5	0	5.287	-1.137	-22%	0.118	2%	1.255	31%
-10	-5	4.809	-20	-2.5	3.727	-5	-10	4.991	-1.081	-22%	0.182	4%	1.264	34%
-35	-35	0.164	-45	-30	0.158	-30	-45	0.132	-0.006	-4%	-0.032	-19%	-0.026	-16%
-17.5	-35	0.219	-22.5	-30	0.247	-15	-45	0.167	0.027	12%	-0.052	-24%	-0.079	-32%
0	-35	0.243	0	-30	0.284	0	-45	0.179	0.041	17%	-0.063	-26%	-0.104	-37%
17.5	-35	0.186	22.5	-30	0.192	15	-45	0.147	0.006	3%	-0.039	-21%	-0.045	-23%
35	-35	0.108	45	-30	0.088	30	-45	0.100	-0.020	-19%	-0.008	-7%	0.012	14%
35	-17.5	0.141	45	-15	0.105	30	-22.5	0.160	-0.036	-25%	0.019	14%	0.055	52%
35	0	0.277	45	0	0.164	30	0	0.401	-0.113	-41%	0.124	45%	0.236	144%
35	17.5	0.503	45	15	0.396	30	22.5	0.466	-0.107	-21%	-0.037	-7%	0.070	18%
35	35	0.254	45	30	0.263	30	45	0.200	0.009	3%	-0.054	-21%	-0.063	-24%
17.5	35	0.315	22.5	30	0.373	15	45	0.234	0.058	18%	-0.081	-26%	-0.139	-37%
0	35	0.417	0	30	0.445	0	45	0.341	0.028	7%	-0.076	-18%	-0.104	-23%
-17.5	35	0.272	-22.5	30	0.286	-15	45	0.214	0.014	5%	-0.057	-21%	-0.071	-25%
-35	35	0.155	-45	30	0.131	-30	45	0.146	-0.024	-15%	-0.009	-6%	0.015	11%
-35	17.5	0.211	-45	15	0.155	-30	22.5	0.232	-0.056	-27%	0.022	10%	0.078	50%
-35	0	0.213	-45	0	0.145	-30	0	0.298	-0.068	-32%	0.084	40%	0.153	106%
-35	-17.5	0.265	-45	-15	0.193	-30	-22.5	0.264	-0.073	-27%	-0.002	-1%	0.071	37%
Standard Deviation:									0.463	15%	0.435	17%	0.747	41%

Table D-2b. Comparisons of Unitized Air Concentrations ($\mu\text{g}/\text{m}^3$ / $\mu\text{g}/\text{s}\cdot\text{m}^2$) for Different Source Shapes and Orientations
(Los Angeles, California)

Source No. 1 (20m x 20m)			Source No. 2 (40m x 10m)			Source No. 3 (10m x 40m)			Differences in UACs Sources No. 1 and No. 2		Differences in UACs Sources No. 1 and No. 3		Differences in UACs Sources No. 2 and No. 3			
Polar Receptor Grid									Diff. In UAC		% of Diff.		Diff. In UAC		% of Diff.	
X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.	Diff. In UAC	% of Diff.		
19	46	0.059	19	46	0.065	19	46	0.069	0.006	9%	0.010	17%	0.005	7%		
38	92	0.016	38	92	0.016	38	92	0.016	0.000	-1%	0.000	3%	0.001	4%		
35	35	0.188	35	35	0.168	35	35	0.284	-0.020	-11%	0.096	51%	0.116	69%		
71	71	0.046	71	71	0.045	71	71	0.052	-0.001	-3%	0.006	13%	0.007	16%		
46	19	0.582	46	19	0.607	46	19	0.461	0.025	4%	-0.121	-21%	-0.146	-24%		
92	38	0.172	92	38	0.174	92	38	0.161	0.003	2%	-0.011	-6%	-0.014	-8%		
50	0	0.278	50	0	0.293	50	0	0.293	0.014	5%	0.015	5%	0.001	0%		
100	0	0.068	100	0	0.067	100	0	0.074	-0.001	-2%	0.005	8%	0.007	10%		
46	-19	0.061	46	-19	0.062	46	-19	0.087	0.002	3%	0.026	43%	0.025	40%		
92	-38	0.015	92	-38	0.015	92	-38	0.016	0.000	0%	0.002	10%	0.002	11%		
35	-35	0.062	35	-35	0.068	35	-35	0.062	0.006	10%	0.000	0%	-0.006	-9%		
71	-71	0.016	71	-71	0.017	71	-71	0.017	0.001	4%	0.001	3%	0.000	-1%		
19	-46	0.080	19	-46	0.076	19	-46	0.087	-0.004	-4%	0.007	9%	0.011	14%		
38	-92	0.023	38	-92	0.022	38	-92	0.024	-0.001	-5%	0.001	3%	0.002	8%		
0	-50	0.086	0	-50	0.084	0	-50	0.096	-0.003	-3%	0.009	11%	0.012	15%		
0	-100	0.023	0	-100	0.024	0	-100	0.024	0.000	1%	0.001	3%	0.000	2%		
-19	-46	0.099	-19	-46	0.092	-19	-46	0.108	-0.006	-7%	0.009	9%	0.016	17%		
-38	-92	0.028	-38	-92	0.027	-38	-92	0.028	-0.001	-2%	0.000	1%	0.001	3%		
-35	-35	0.122	-35	-35	0.119	-35	-35	0.143	-0.003	-2%	0.021	18%	0.024	20%		
-71	-71	0.033	-71	-71	0.032	-71	-71	0.034	0.000	-1%	0.001	4%	0.002	5%		
-46	-19	0.218	-46	-19	0.223	-46	-19	0.226	0.005	2%	0.008	4%	0.003	2%		
-92	-38	0.060	-92	-38	0.061	-92	-38	0.061	0.001	1%	0.001	1%	0.000	0%		
-50	0	0.320	-50	0	0.378	-50	0	0.278	0.057	18%	-0.042	-13%	-0.099	-26%		
-100	0	0.093	-100	0	0.098	-100	0	0.087	0.005	6%	-0.006	-6%	-0.011	-11%		
-46	19	0.264	-46	19	0.273	-46	19	0.260	0.009	3%	-0.005	-2%	-0.013	-5%		
-92	38	0.074	-92	38	0.075	-92	38	0.073	0.001	1%	-0.001	-2%	-0.002	-2%		
-35	35	0.137	-35	35	0.123	-35	35	0.164	-0.014	-10%	0.027	20%	0.041	33%		
-71	71	0.037	-71	71	0.035	-71	71	0.039	-0.002	-5%	0.002	4%	0.003	9%		
-19	46	0.063	-19	46	0.066	-19	46	0.073	0.003	4%	0.010	15%	0.007	11%		
-38	92	0.017	-38	92	0.017	-38	92	0.018	0.000	-2%	0.001	3%	0.001	5%		
0	50	0.067	0	50	0.058	0	50	0.080	-0.008	-12%	0.014	21%	0.022	37%		
0	100	0.020	0	100	0.018	0	100	0.021	-0.002	-9%	0.001	6%	0.003	15%		
Standard Deviation:									0.013	6%	0.030	14%	0.040	18%		

**Table D-2b. Comparisons of Unitized Air Concentrations ($\mu\text{g}/\text{m}^3$ / $\mu\text{g}/\text{s}\cdot\text{m}^2$) for Different Source Shapes and Orientations
(Los Angeles, California)**

Source No. 1 (20m x 20m)			Source No. 2 (40m x 10m)			Source No. 3 (10m x 40m)			Differences in UACs Sources No. 1 and No. 2		Differences in UACs Sources No. 1 and No. 3		Differences in UACs Sources No. 2 and No. 3	
Cartesian Receptor Grid									Diff. In UAC % of Diff.		Diff. In UAC % of Diff.		Diff. In UAC % of Diff.	
X (m)	Y (m)	UAC	X (m)	Y (m)	UAC	X (m)	Y (m)	UAC						
-10	-10	3.225	-20	-5	3.241	-5	-20	2.674	0.016	1%	-0.551	-17%	-0.567	-17%
-5	-10	4.025	-10	-5	4.333	-2.5	-20	3.119	0.308	8%	-0.906	-23%	-1.214	-28%
0	-10	3.952	0	-5	4.297	0	-20	3.050	0.345	9%	-0.902	-23%	-1.247	-29%
5	-10	3.431	10	-5	3.871	2.5	-20	2.564	0.440	13%	-0.867	-25%	-1.307	-34%
10	-10	1.683	20	-5	1.592	5	-20	1.511	-0.091	-5%	-0.172	-10%	-0.081	-5%
10	-5	5.931	20	-2.5	4.787	5	-10	5.570	-1.143	-19%	-0.360	-6%	0.783	16%
10	0	6.636	20	0	5.882	5	0	5.644	-0.754	-11%	-0.992	-15%	-0.238	-4%
10	5	6.640	20	2.5	6.294	5	10	5.524	-0.346	-5%	-1.116	-17%	-0.770	-12%
10	10	5.600	20	5	5.866	5	20	4.325	0.266	5%	-1.275	-23%	-1.541	-26%
5	10	6.893	10	5	8.126	2.5	20	4.939	1.232	18%	-1.955	-28%	-3.187	-39%
0	10	6.860	0	5	8.285	0	20	4.913	1.424	21%	-1.947	-28%	-3.371	-41%
-5	10	6.031	-10	5	7.442	-2.5	20	4.156	1.411	23%	-1.875	-31%	-3.286	-44%
-10	10	3.393	-20	5	3.497	-5	20	2.702	0.103	3%	-0.691	-20%	-0.794	-23%
-10	5	5.649	-20	2.5	5.102	-5	10	5.015	-0.547	-10%	-0.634	-11%	-0.088	-2%
-10	0	5.944	-20	0	5.373	-5	0	5.167	-0.572	-10%	-0.777	-13%	-0.205	-4%
-10	-5	5.663	-20	-2.5	5.028	-5	-10	5.104	-0.635	-11%	-0.559	-10%	0.076	2%
-35	-35	0.124	-45	-30	0.139	-30	-45	0.095	0.014	11%	-0.029	-23%	-0.043	-31%
-17.5	-35	0.158	-22.5	-30	0.183	-15	-45	0.123	0.025	16%	-0.035	-22%	-0.060	-33%
0	-35	0.172	0	-30	0.199	0	-45	0.121	0.028	16%	-0.050	-29%	-0.078	-39%
17.5	-35	0.123	22.5	-30	0.124	15	-45	0.100	0.001	0%	-0.024	-19%	-0.024	-20%
35	-35	0.064	45	-30	0.053	30	-45	0.063	-0.011	-17%	-0.001	-2%	0.010	19%
35	-17.5	0.095	45	-15	0.076	30	-22.5	0.119	-0.019	-20%	0.024	25%	0.043	57%
35	0	0.592	45	0	0.377	30	0	0.696	-0.215	-36%	0.104	18%	0.319	85%
35	17.5	0.829	45	15	0.739	30	22.5	0.683	-0.090	-11%	-0.146	-18%	-0.055	-7%
35	35	0.192	45	30	0.304	30	45	0.101	0.112	58%	-0.091	-47%	-0.203	-67%
17.5	35	0.109	22.5	30	0.195	15	45	0.072	0.086	78%	-0.037	-34%	-0.122	-63%
0	35	0.125	0	30	0.144	0	45	0.100	0.019	15%	-0.025	-20%	-0.044	-31%
-17.5	35	0.113	-22.5	30	0.160	-15	45	0.077	0.047	42%	-0.035	-31%	-0.082	-52%
-35	35	0.139	-45	30	0.166	-30	45	0.089	0.026	19%	-0.050	-36%	-0.077	-46%
-35	17.5	0.387	-45	15	0.335	-30	22.5	0.370	-0.053	-14%	-0.017	-4%	0.036	11%
-35	0	0.603	-45	0	0.472	-30	0	0.603	-0.131	-22%	0.000	0%	0.131	28%
-35	-17.5	0.318	-45	-15	0.275	-30	-22.5	0.316	-0.043	-13%	-0.002	-1%	0.041	15%
Standard Deviation:									0.542	24%	0.614	15%	1.026	33%

D.3 Receptor Locations and Spacings

A sensitivity analysis was conducted using the ISCST3 model to determine what receptor locations and spacings should be used in the risk analysis for five types of waste management units (WMUs). A discussion of the analysis follows.

Because it takes a substantial amount of time for the ISCST3 model to execute, it was necessary to choose a limited number of receptors to be used in the dispersion modeling analysis. The larger the number of receptor points, the longer the run time. However, modeling fewer receptors may result in the omission of the maximum point for assessing exposure impacts. Therefore, a sensitivity analysis was conducted to determine the number of receptors needed for the model run and to locate ideal receptor placements.

A wind rose was plotted for each of the 29 meteorological stations to be used in the risk analysis for a 5-year time period in order to choose two meteorological stations for this sensitivity analysis. Little Rock, Arkansas, and Los Angeles, California, meteorological stations were selected for the sensitivity analysis. The wind roses show that Little Rock has very evenly distributed wind directions, and Los Angeles has a predominant southwest to west wind (Figure D-3). Little Rock and Los Angeles were chosen to determine if a higher density of receptors should be placed downwind of a site near Los Angeles, as compared to a site near Little Rock. Similarly, the 5th, 50th, and 95th percentile of sizes of LAUs were used in the sensitivity analysis to determine whether sizes of units can affect receptor locations and spacings. The areas of the 5th, 50th, and 95th percentile of sizes of LAUs are 1,200 m², 100,000 m², and 1,700,000 m², respectively.

The dispersion modeling was conducted using two sets of receptor grids. The first set of receptor points (Cartesian receptor grid) was placed around the modeled source with distances of 0, 25, 50, 75, and 150 meters from the edge of the unit. Square-shaped ground-level area sources were used in the modeling. Therefore, these receptors are located on five squares surrounding the source. The second set of receptor points (polar receptor grid) was placed outside of the first set of receptors to 10 kilometers from the center of the source. Since the ISCST3 model's area source algorithm does not consider elevated terrain, receptor elevations were not input in the modeling.

In this sensitivity analysis, both downwind and lateral receptor spacings were investigated for three unit sizes using 5 years of meteorological data from Little Rock and Los Angeles. For the first set of receptor points (i.e., Cartesian receptor grid), five downwind distances of 0, 25, 50, 75, and 150 meters from the edge of the source were used. For lateral receptor spacing, choices of 64, 32, and 16 equally spaced receptor points for each square were used in the modeling to determine the number of receptors needed to catch the maximum impacts. (See Figures D-5a through D-5c for Cartesian receptor locations and spacings [50th percentile]). For the second set of receptor points (i.e., polar receptor grid), about 20 downwind distances (i.e., receptor rings) were used. Receptor lateral intervals of 22.5° and 10° were used to determine whether 22.5° spacing can catch the maximum impacts. With a 22.5° interval, there are 16 receptors on each ring. There are 36 receptors on each ring for the 10° interval. See Figures D-6a and D-6b for polar receptor locations (5th percentile).

The results (Figures D-7a through D-7f) show that the maximum downwind concentrations decrease sharply from the edge of the area source to 150 meters from the source. The maximum concentrations decrease more sharply for a smaller area source than for a larger one. This means that more close-to-source receptors are generally needed for a small area source than for a large one.

The results also show that the maximum impacts are generally higher for a dense receptor grid (i.e., 64 or 32 receptors on each square) than for a scattered receptor grid (i.e., 16 receptors on each square). However, the differences of the maximum receptor impacts are not significant between a dense and a scattered receptor grid (Figures D-7a through D-7f). It should be noted that the above conclusions apply to both Little Rock and Los Angeles. This means that the distribution of wind directions does not play an important role in determining receptor lateral spacings.

Figures D-8a through D-8f compare the maximum concentrations at each ring for 22.5° and 10° intervals. The results show that the differences of the maximum concentrations are greater for close-to-source receptors than for further out receptors, and the differences are greater for larger area sources than for smaller area sources. The differences of the maximum concentrations for 22.5° and 10° intervals are generally small, and the concentrations tend to be the same at 10 kilometers. The conclusions were drawn from both Little Rock and Los Angeles meteorological data.

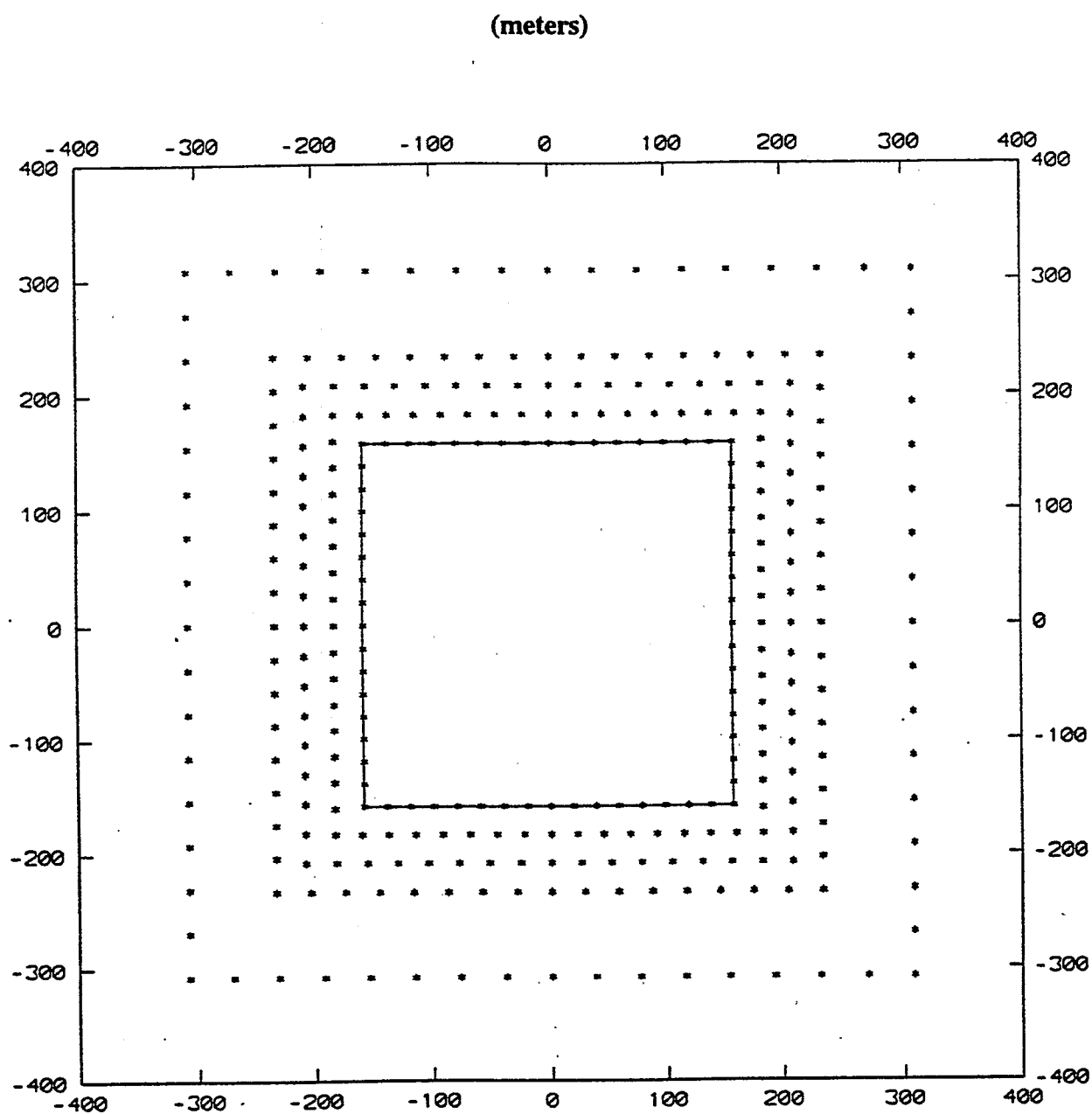


Figure D-5a. Cartesian Receptor Grid (50th percentile, 64 receptors each square)

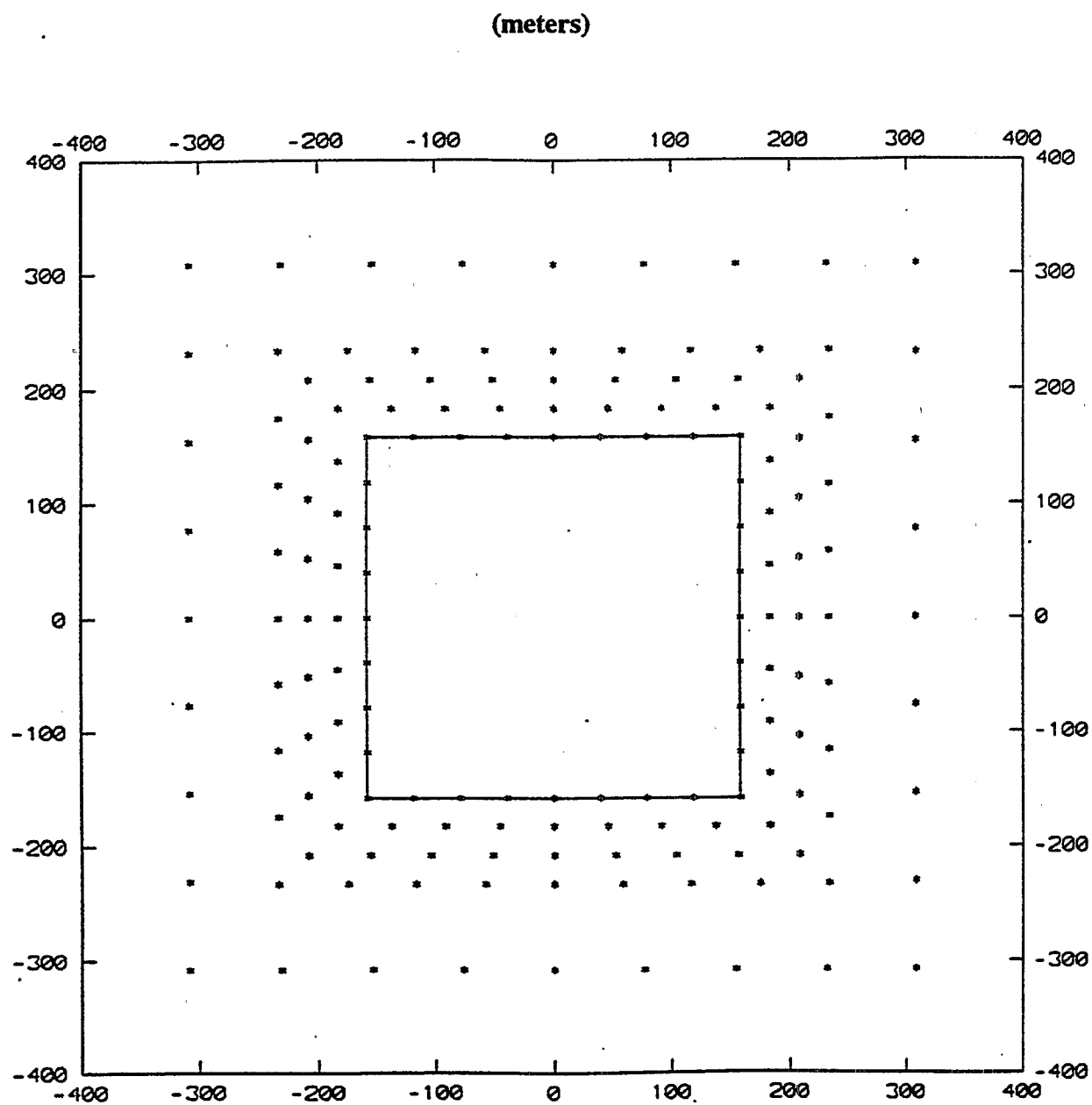


Figure D-5b. Cartesian Receptor Grid (50th percentile, 32 receptors each square)

(meters)

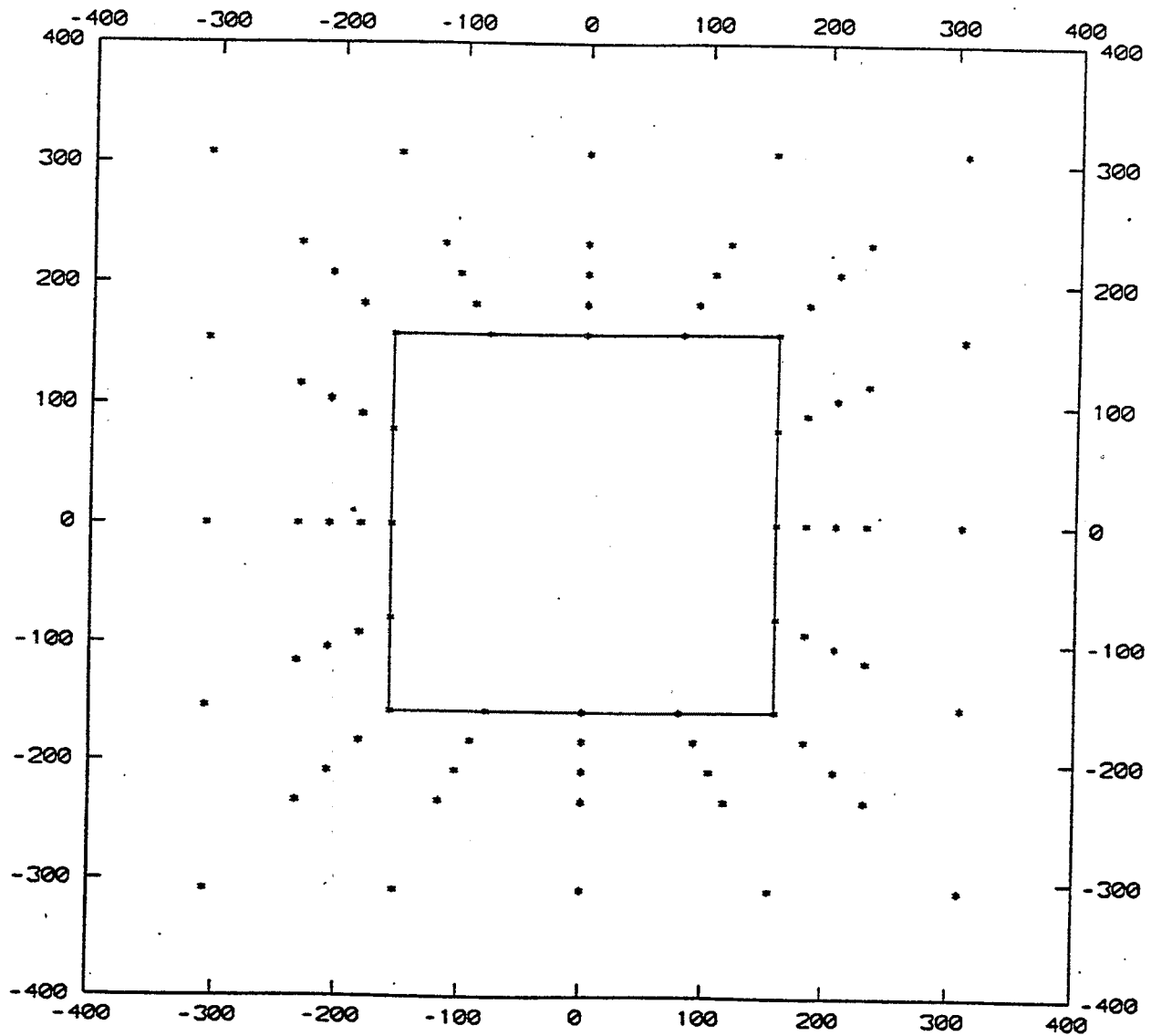


Figure D-5c. Cartesian Receptor Grid (50th percentile, 16 receptors each square)

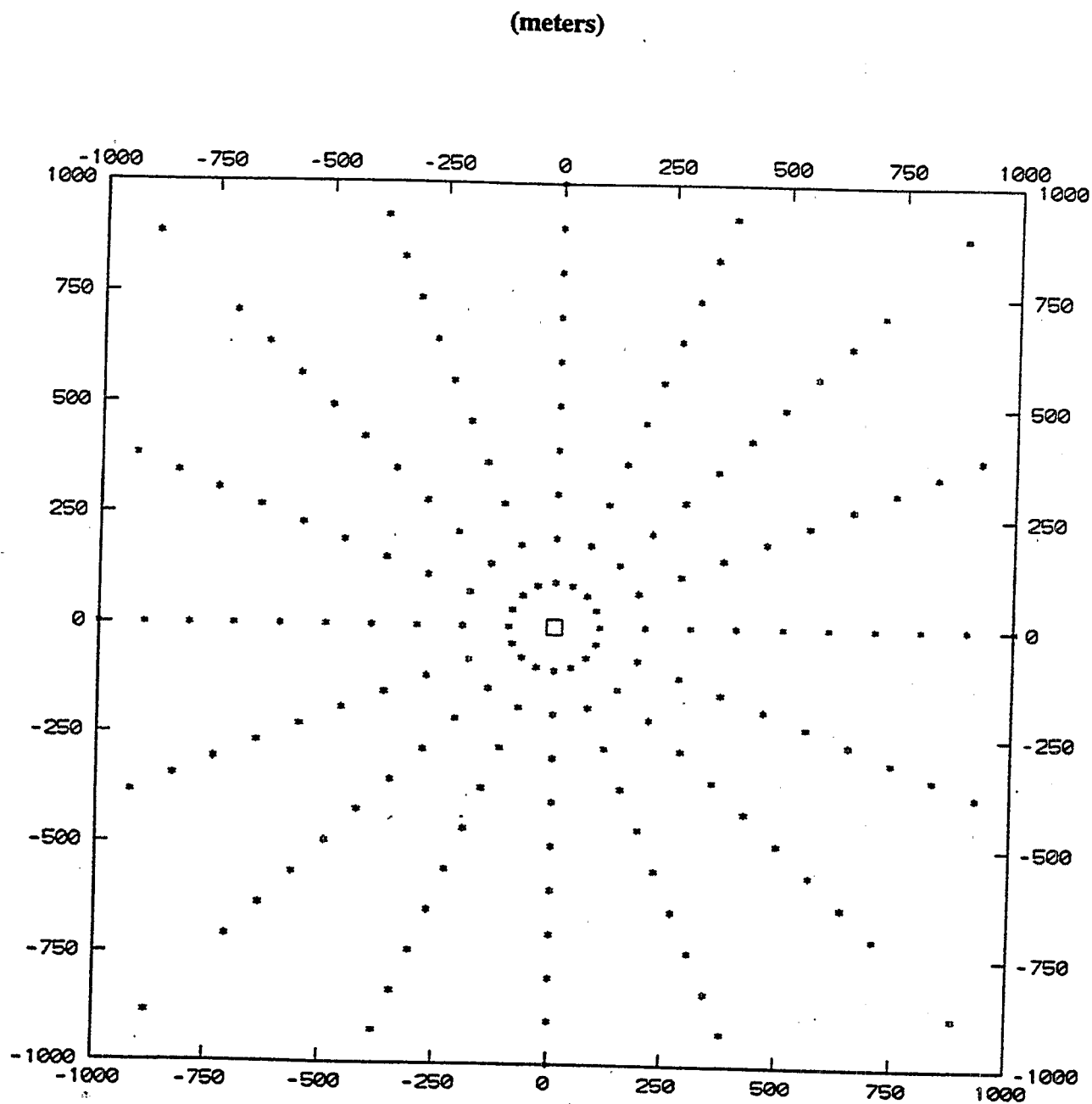


Figure D-6a. Polar Receptor Grid (22.5 degree, 5th percentile)

(meters)

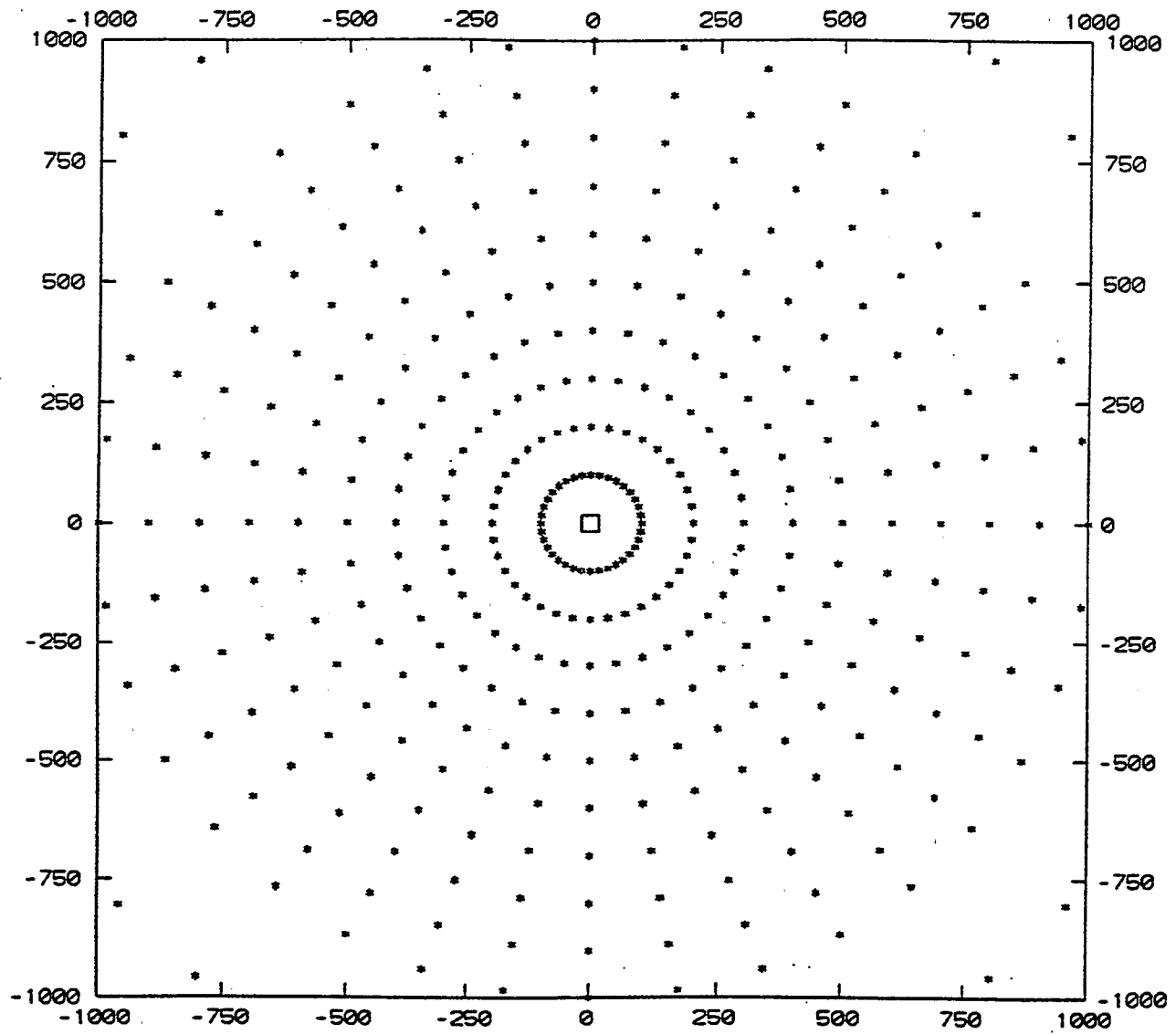
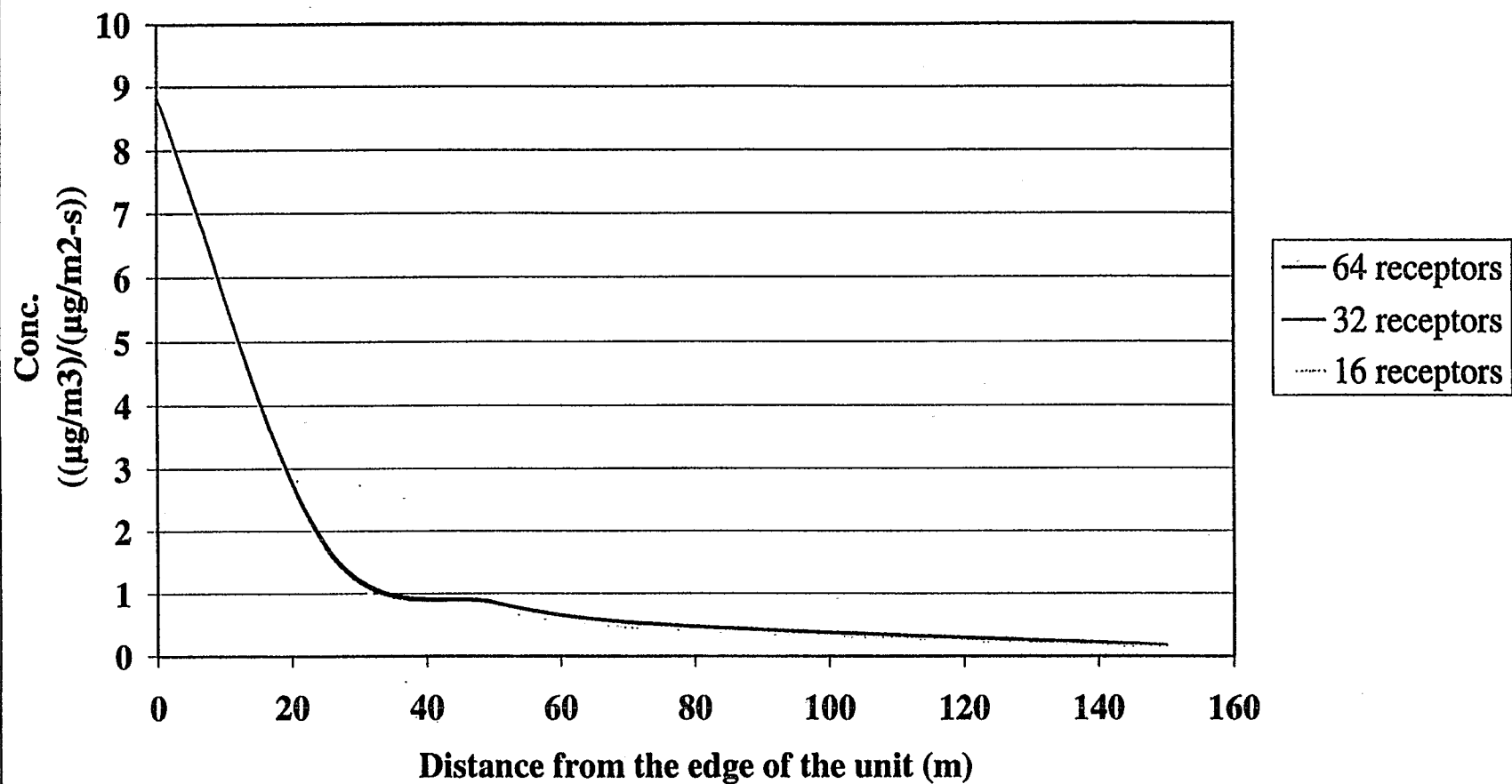
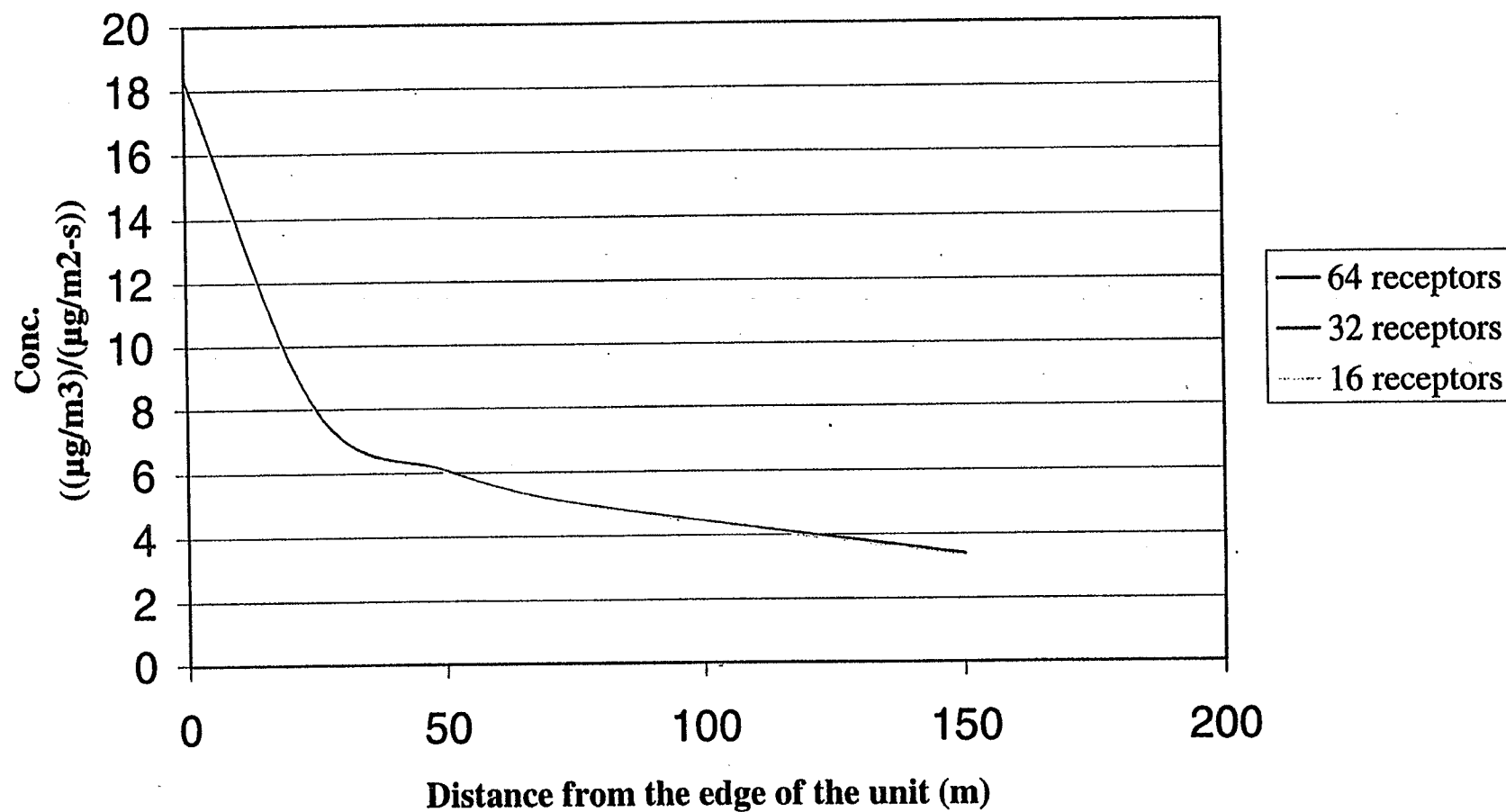


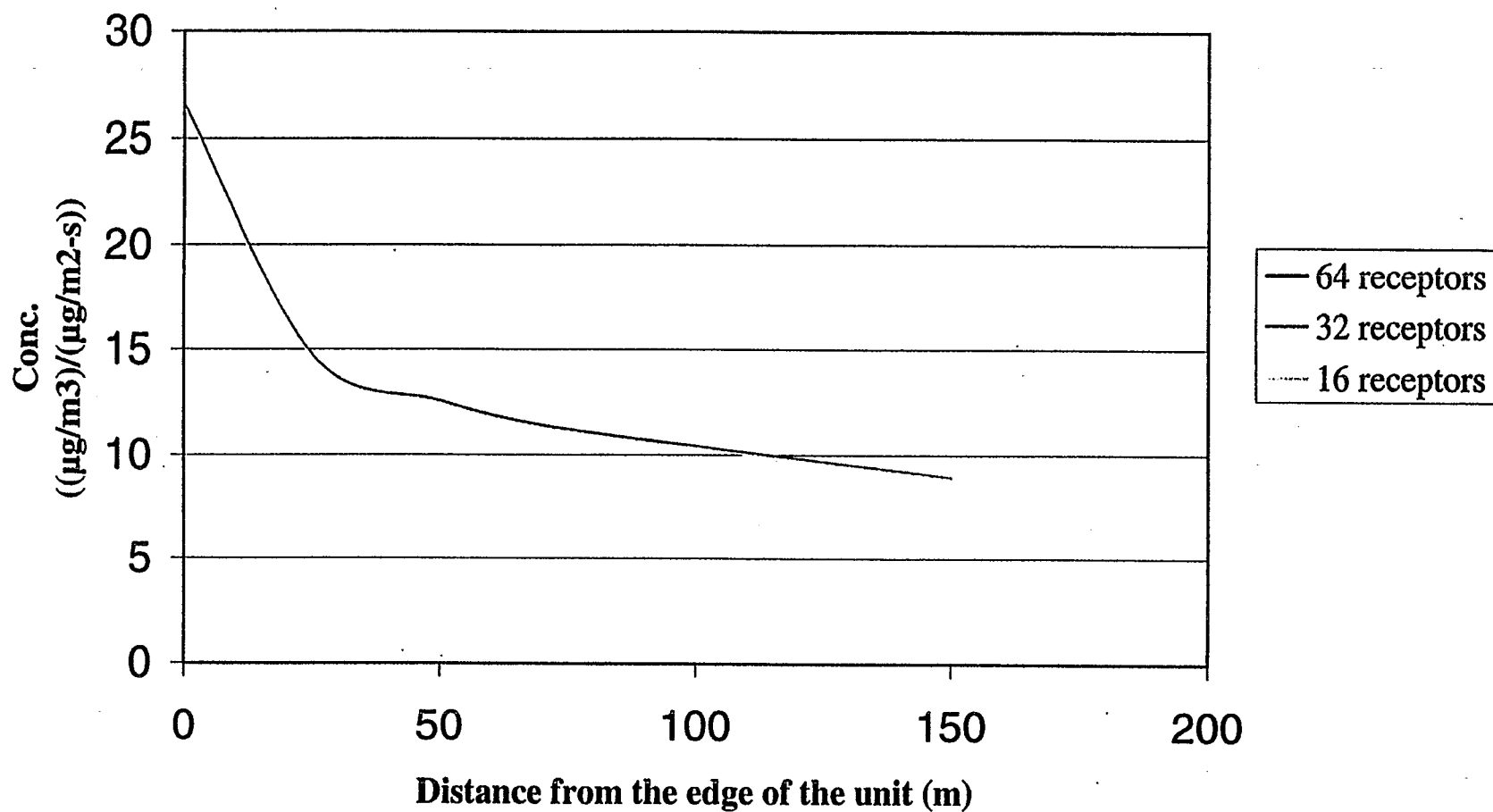
Figure D-6b. Polar Receptor Grid (10 degree, 5th percentile)



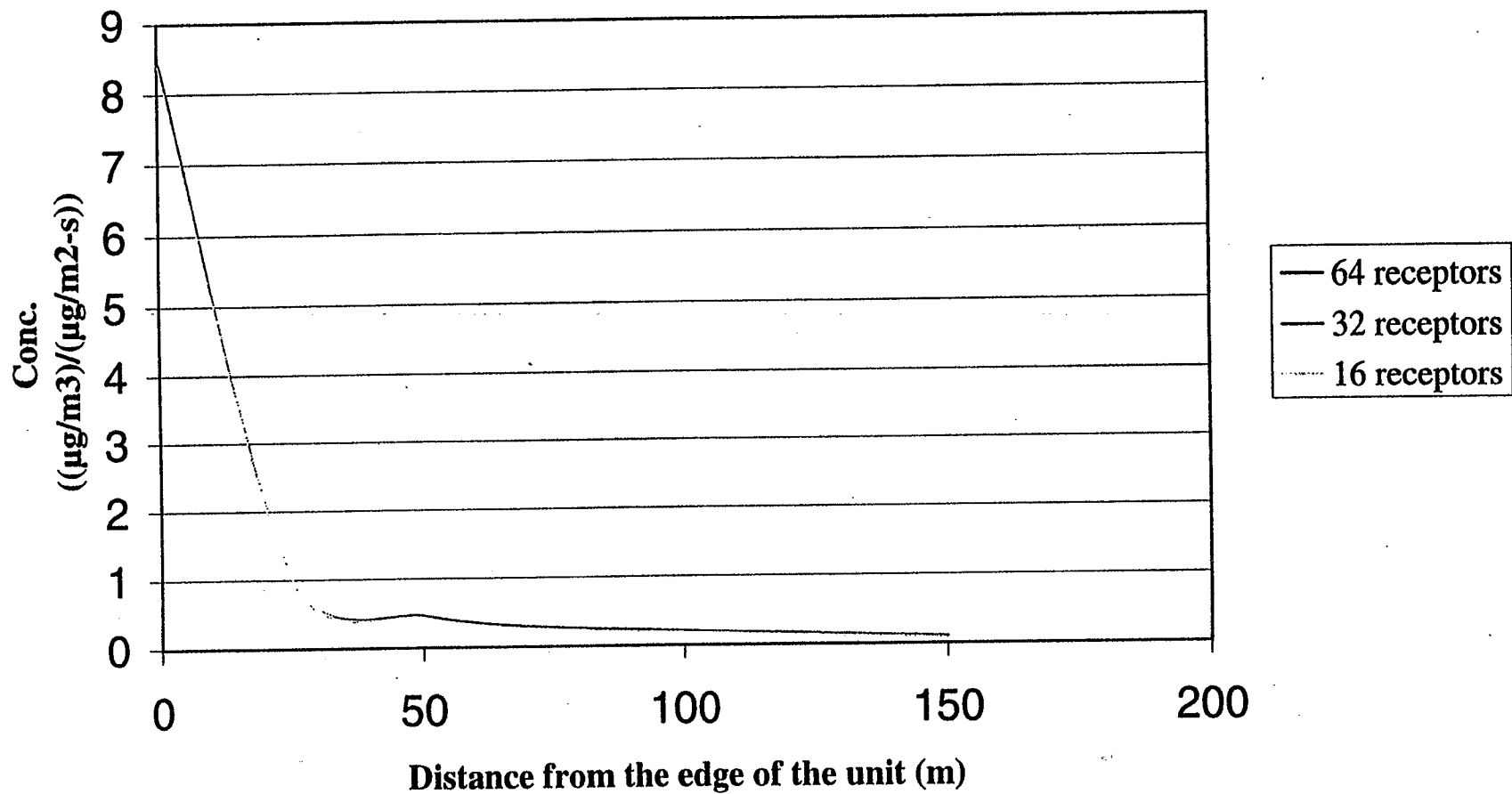
**Figure D-7a. Maximum Concentrations
(5th Percentile, LAU, Los Angeles, CA)**



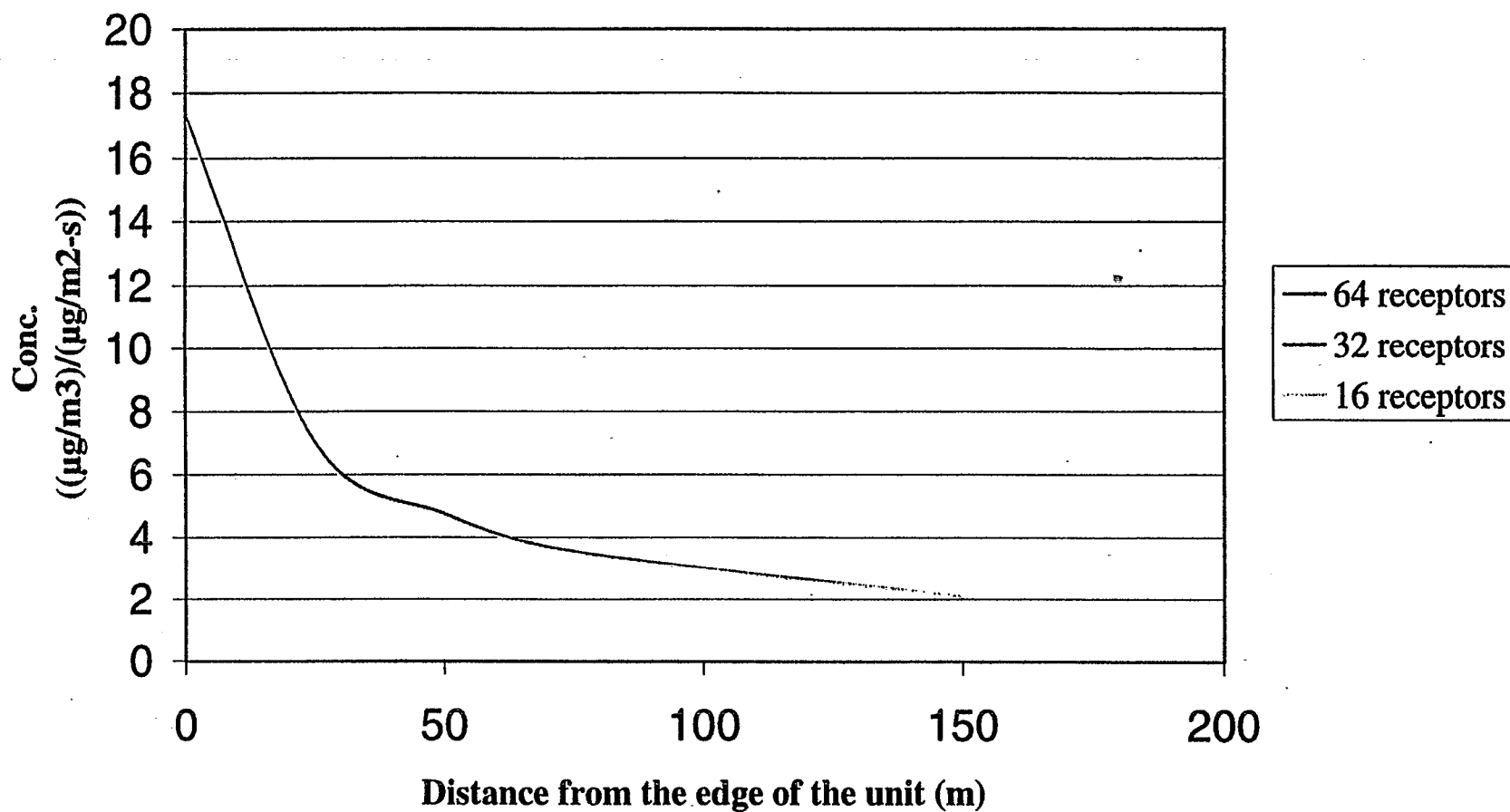
**Figure D-7b. Maximum Concentrations
(50th Percentile, LAU, Los Angeles, CA)**



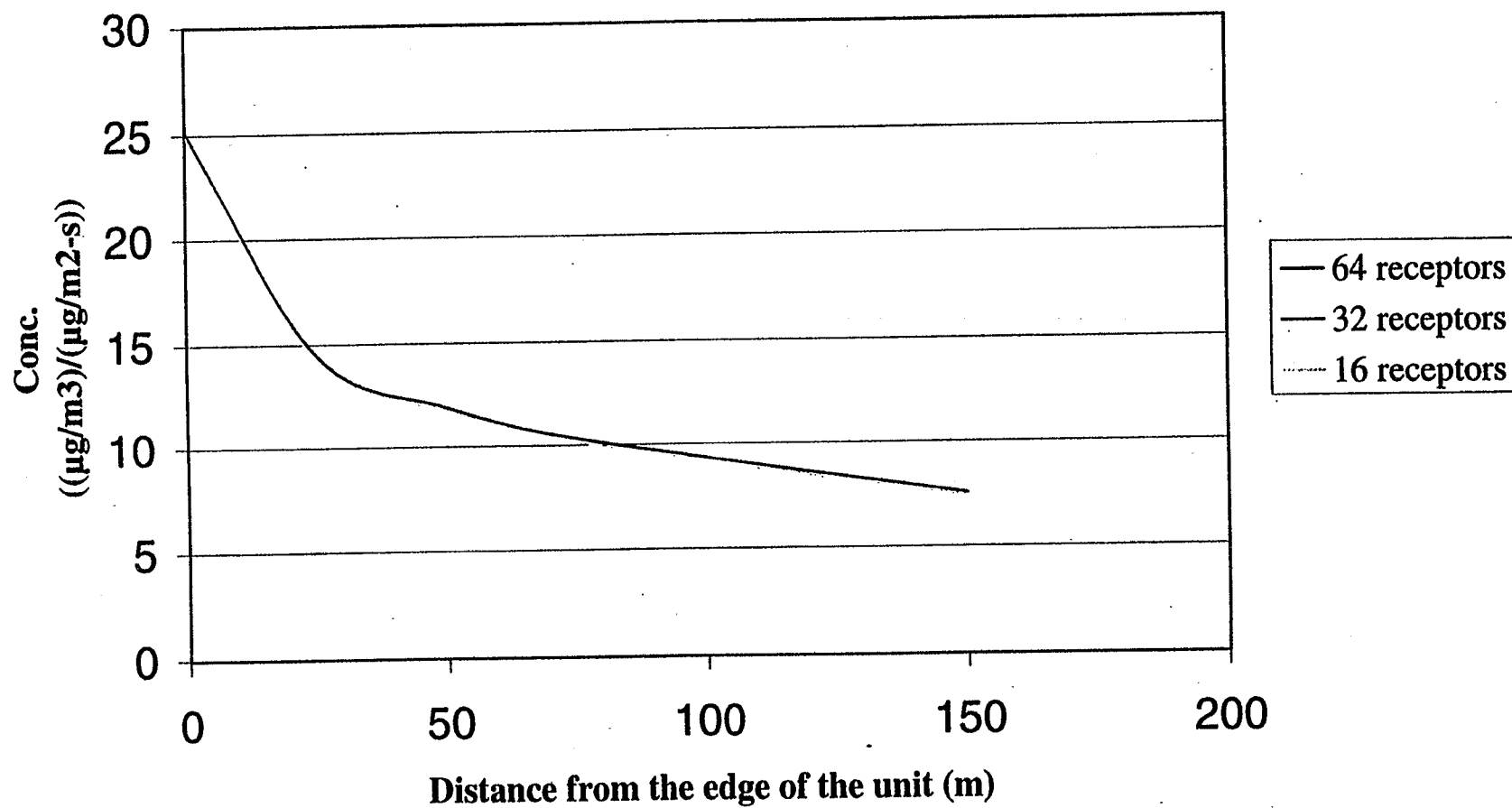
**Figure D-7c. Maximum Concentrations
(95th Percentile, LAU, Los Angeles, CA)**



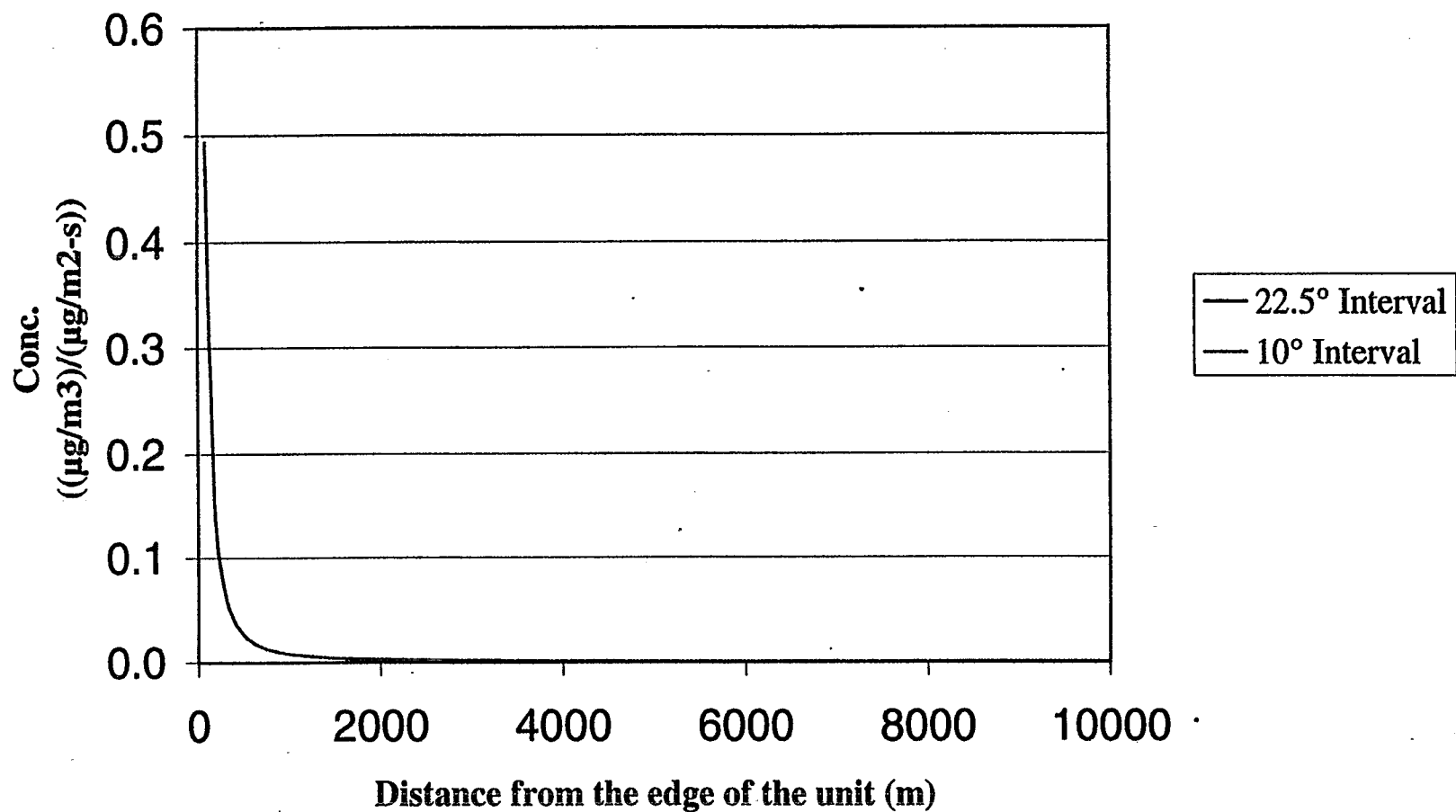
**Figure D-7d. Maximum Concentrations
(5th Percentile, LAU, Little Rock, AR)**



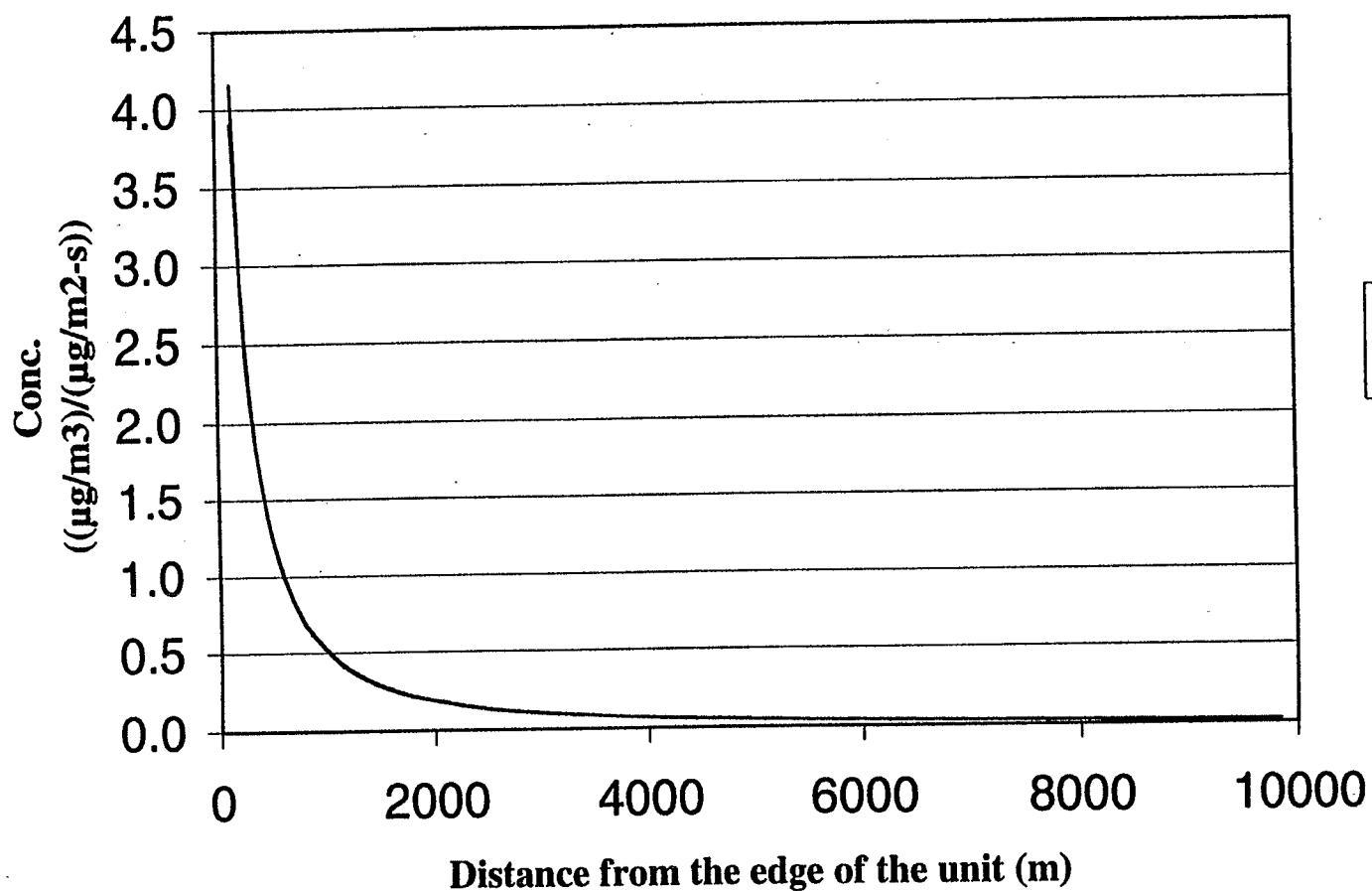
**Figure D-7e. Maximum Concentrations
(50th Percentile, LAU, Little Rock, AR)**



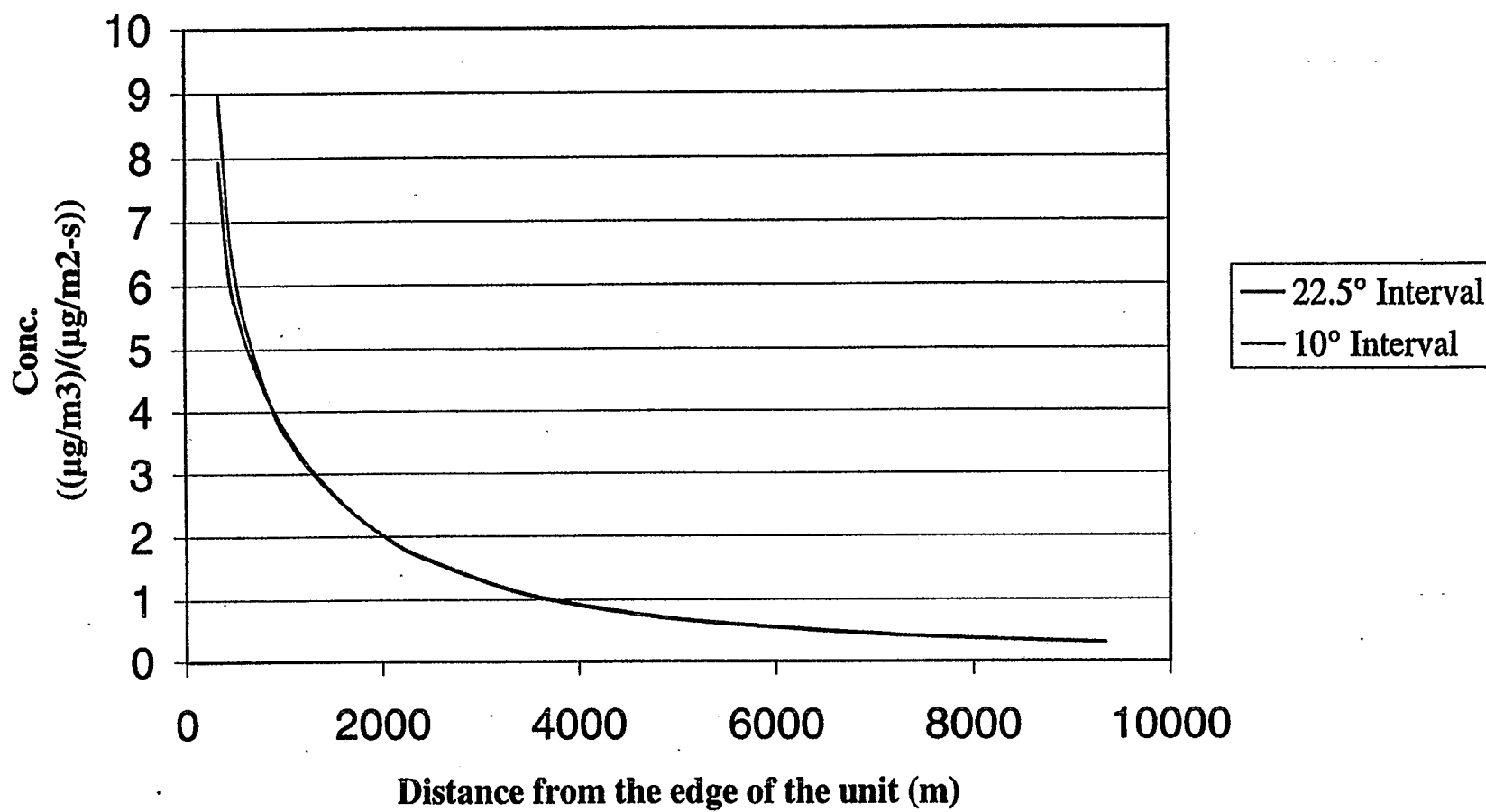
**Figure D-7f. Maximum Concentrations
(95th Percentile, LAU, Little Rock, AR)**



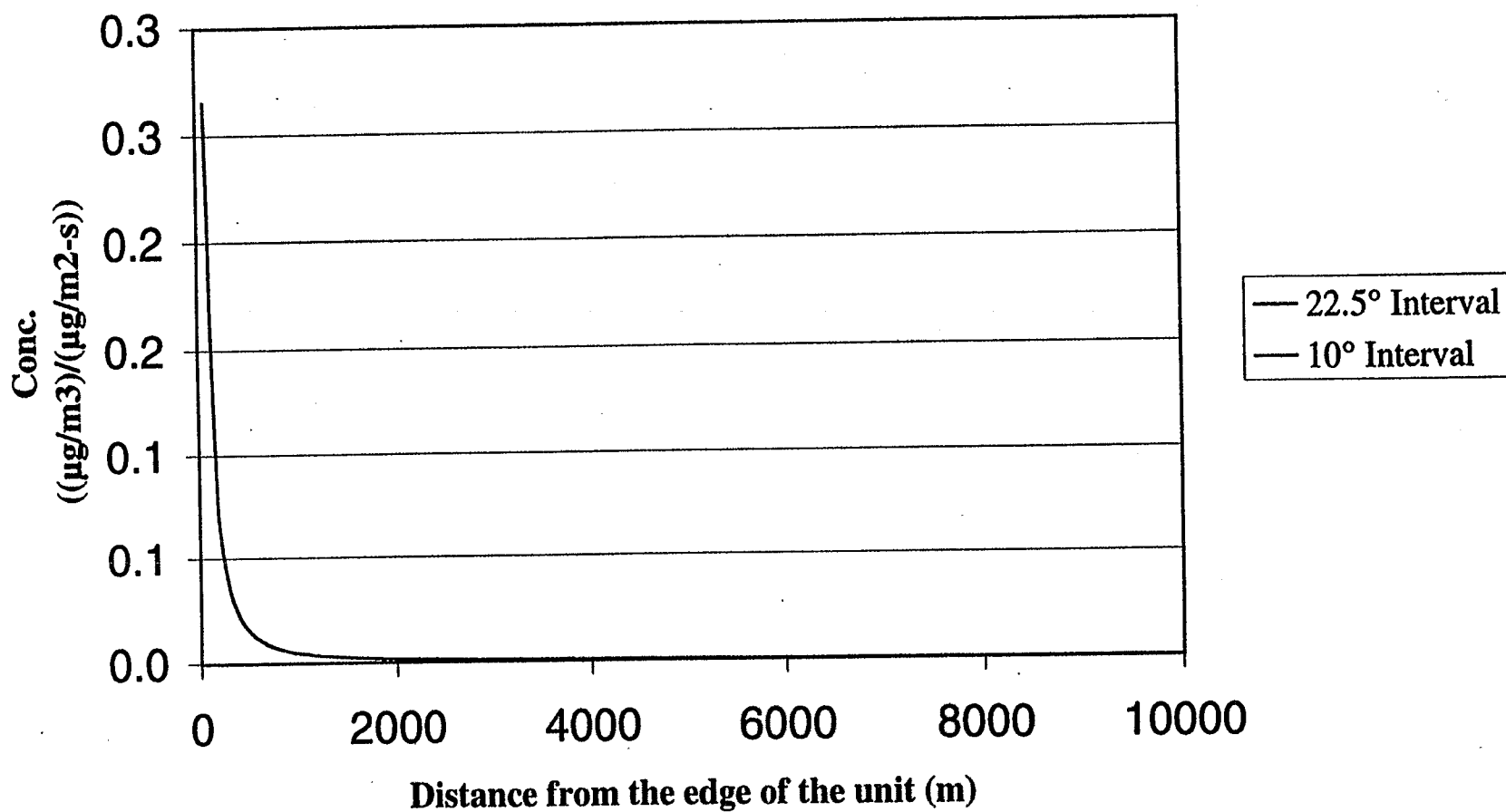
**Figure D-8a. Maximum Concentrations
(5th Percentile, LAU, Los Angeles, CA)**



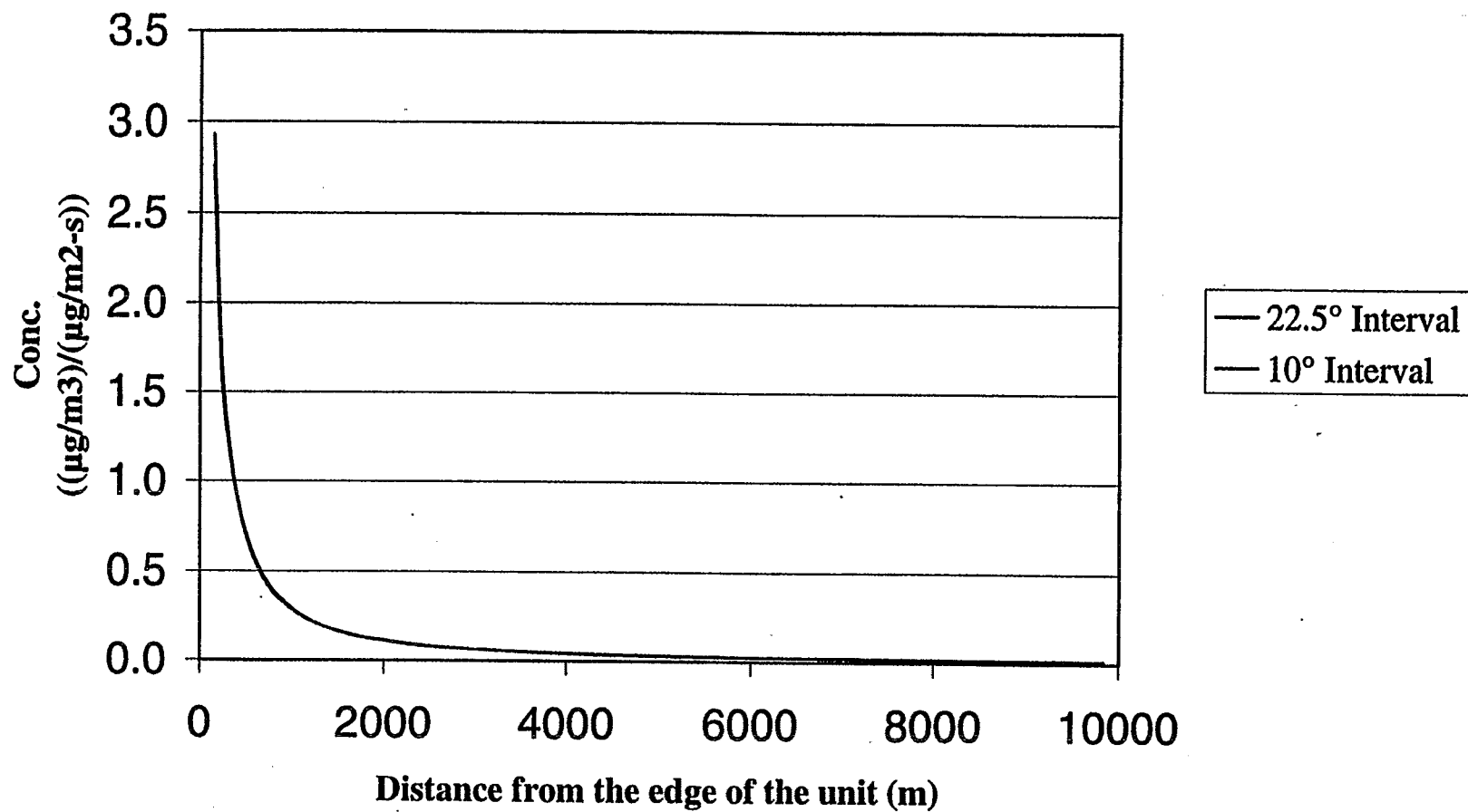
**Figure D-8b. Maximum Concentrations
(50th Percentile, LAU, Los Angeles, CA)**



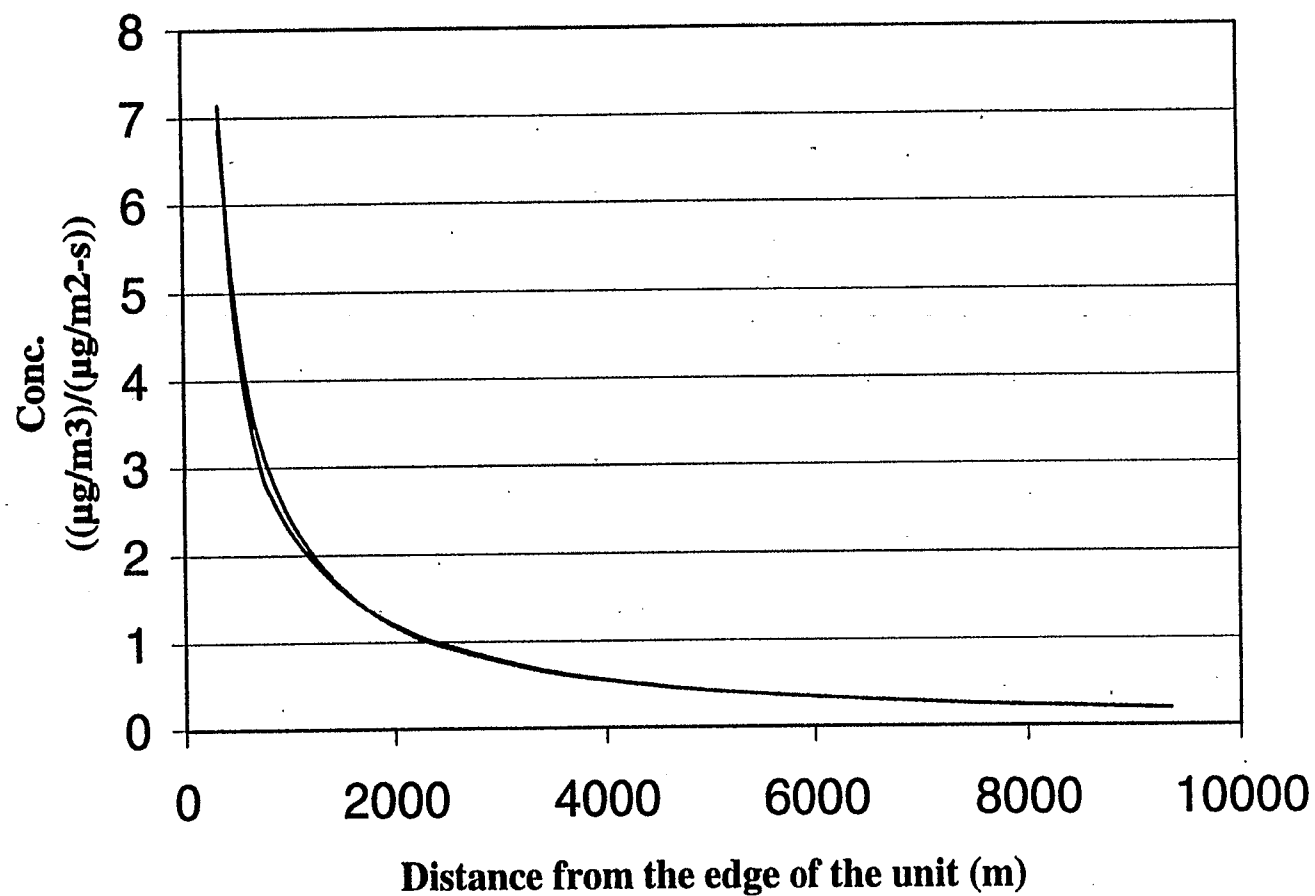
**Figure D-8C. Maximum Concentrations
(95th Percentile, LAU, Los Angeles, CA)**



**Figure D-8d. Maximum Concentrations
(5th Percentile, LAU, Little Rock, AR)**



**Figure D-8e. Maximum Concentrations
(50th Percentile, LAU, Little Rock, AR)**



**Figure D-8f. Maximum Concentrations
(95th Percentile, LAU, Little Rock, AR)**

D.4 An Analysis on Windroses at the 29 Sites

The hourly meteorological data from the 29 meteorological stations used in the Air Characteristic Study were used to generate windroses. A windrose consists of 16 directions, with the angle between any two adjacent directions being 22.5° . The prevailing wind directions for the 29 meteorological stations were counted to estimate the number of entries in each wind directions category. The results are presented in Figure D-9.

The narrowness of the most frequent wind directions for each of the 29 meteorological stations was examined. Based on the narrowness (or angles) of the most frequent wind directions, four categories were used to distinguish the windroses for the 29 meteorological stations. The four categories of windroses are:

- Narrowly distributed: most frequent wind directions no greater than 45°
- Moderately distributed: most frequent wind directions no greater than 90°
- Evenly distributed: no obvious predominant wind directions
- Bimodally distributed: most frequent wind directions are from two opposite directions.

The number of meteorological stations in each category is given in Table D-3. Figure D-10 gives some examples of windroses for each category. The windroses for the 29 meteorological stations are available and can be provided upon request.

An examination of the windroses and the maximum unitized annual average air concentrations from the Air Characteristic Study revealed that the sites with high concentrations are those with narrowly distributed wind directions. Simply put, persistent wind direction consistently blows pollutants from the source to the same receptors. Therefore, the more often the wind blows in a certain direction, the more likely high cumulative concentrations will occur at sites in that direction.

Air concentrations from a source are inversely proportion to windspeed. Given the same distribution of wind directions, a site with lower windspeed will have higher concentrations. The windroses show that, in the prevailing wind direction, the percentage of light wind occurring at a site with narrowly distributed wind directions is often higher than that at a site with evenly distributed wind directions. Therefore, we can conclude that a site with narrowly distributed wind directions will most likely produce the highest long-term average air concentrations.

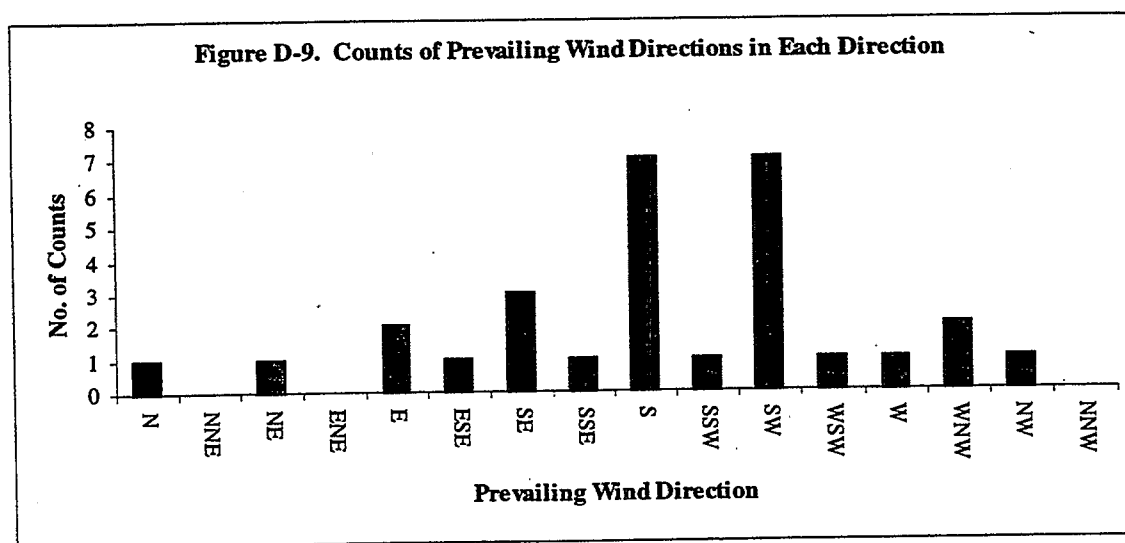
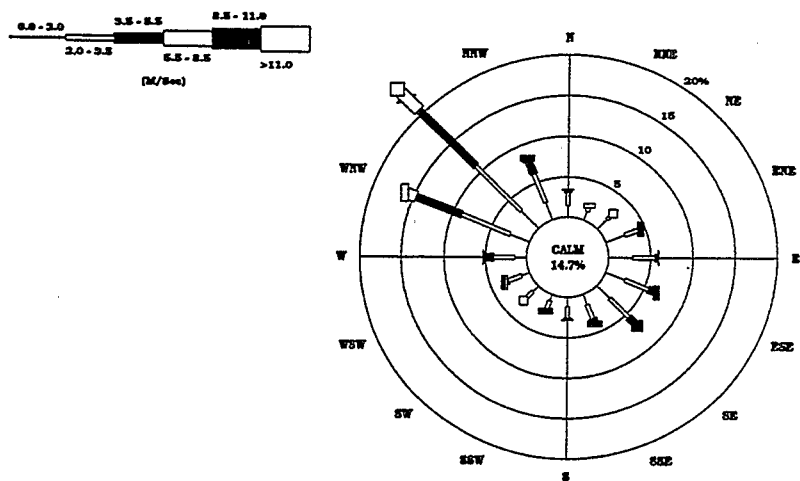


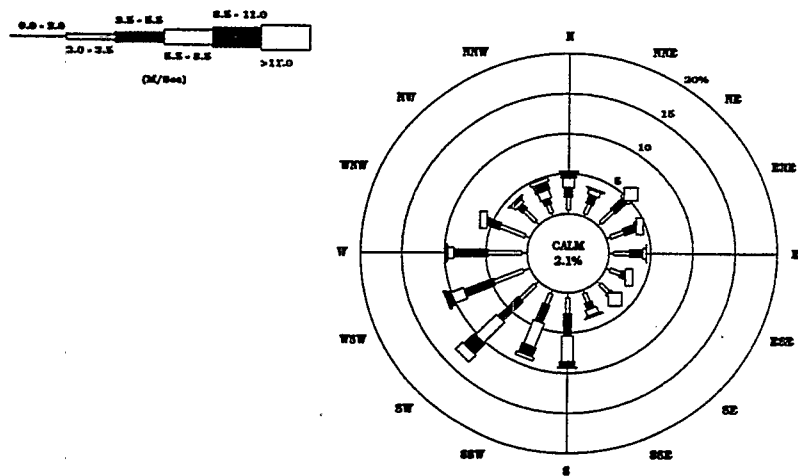
Table D-3. No. of Met Stations with Different Shapes of Windroses

Shape of Windrose	No. of Stations
Narrowly distributed	10
Moderately distributed	4
Evenly distributed	6
Bi-modally distributed	9

Figure D-10. Examples of Different Shapes of Windroses

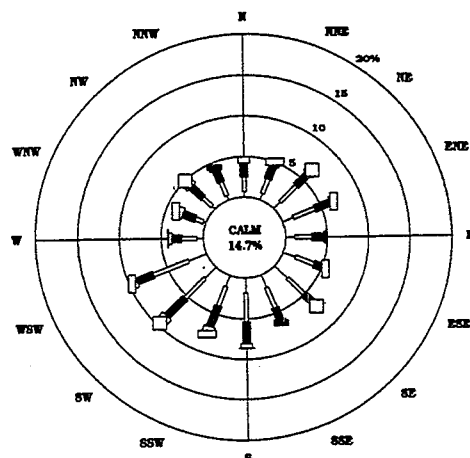
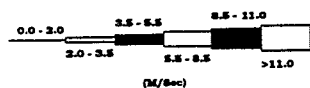


Narrowly Distributed Windrose

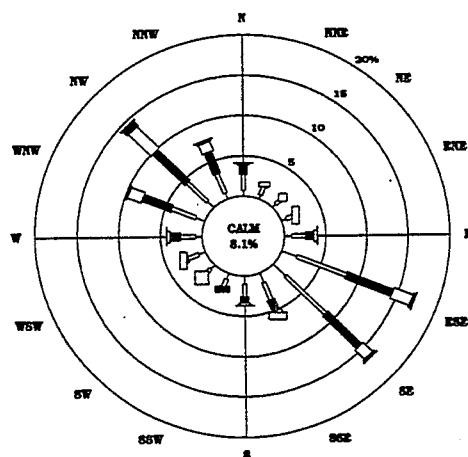
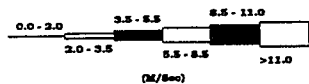


Moderately Distributed Windroses

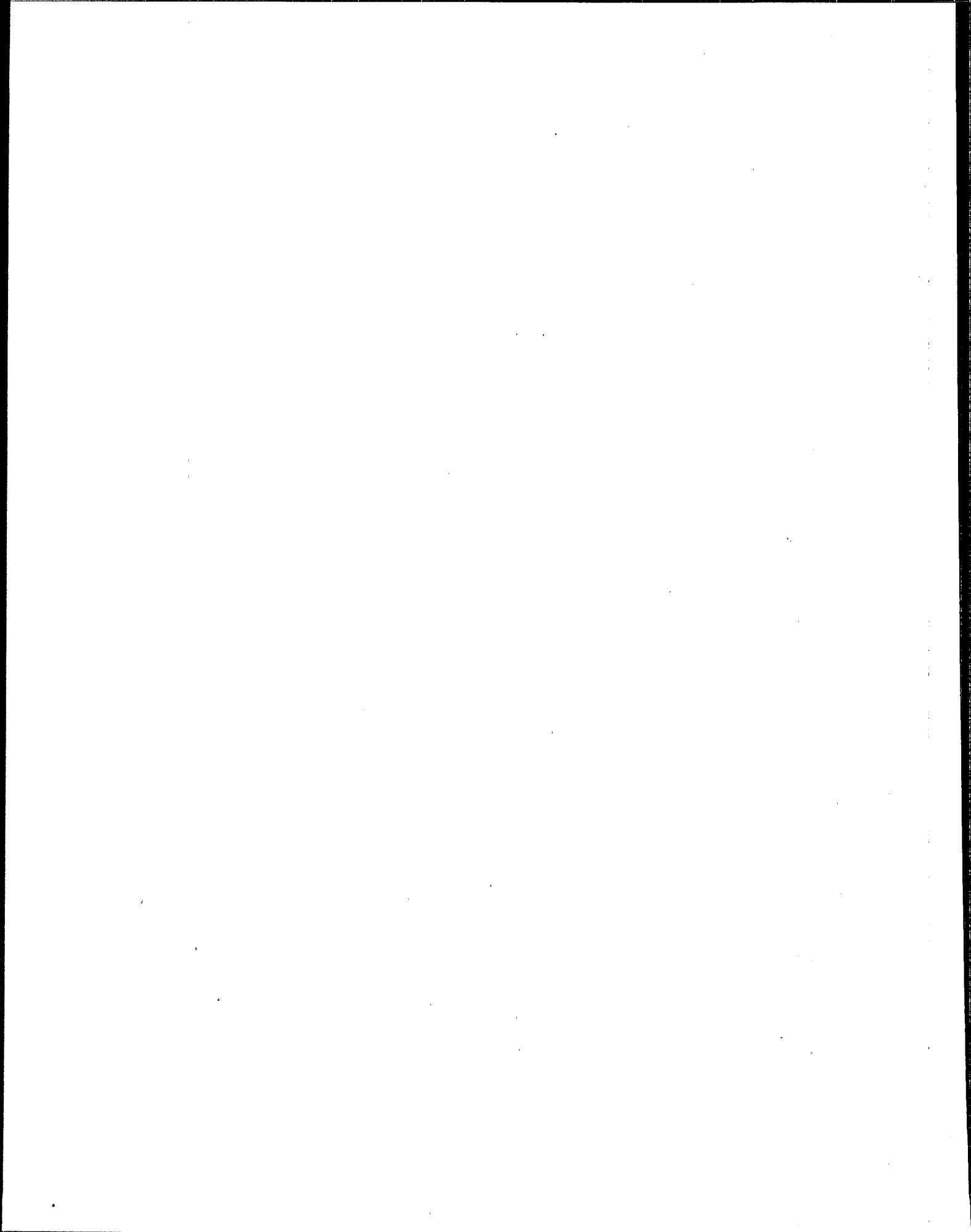
Figure D-10 (Continued). Examples of Different Shapes of Windroses



Evenly Distributed Windrose

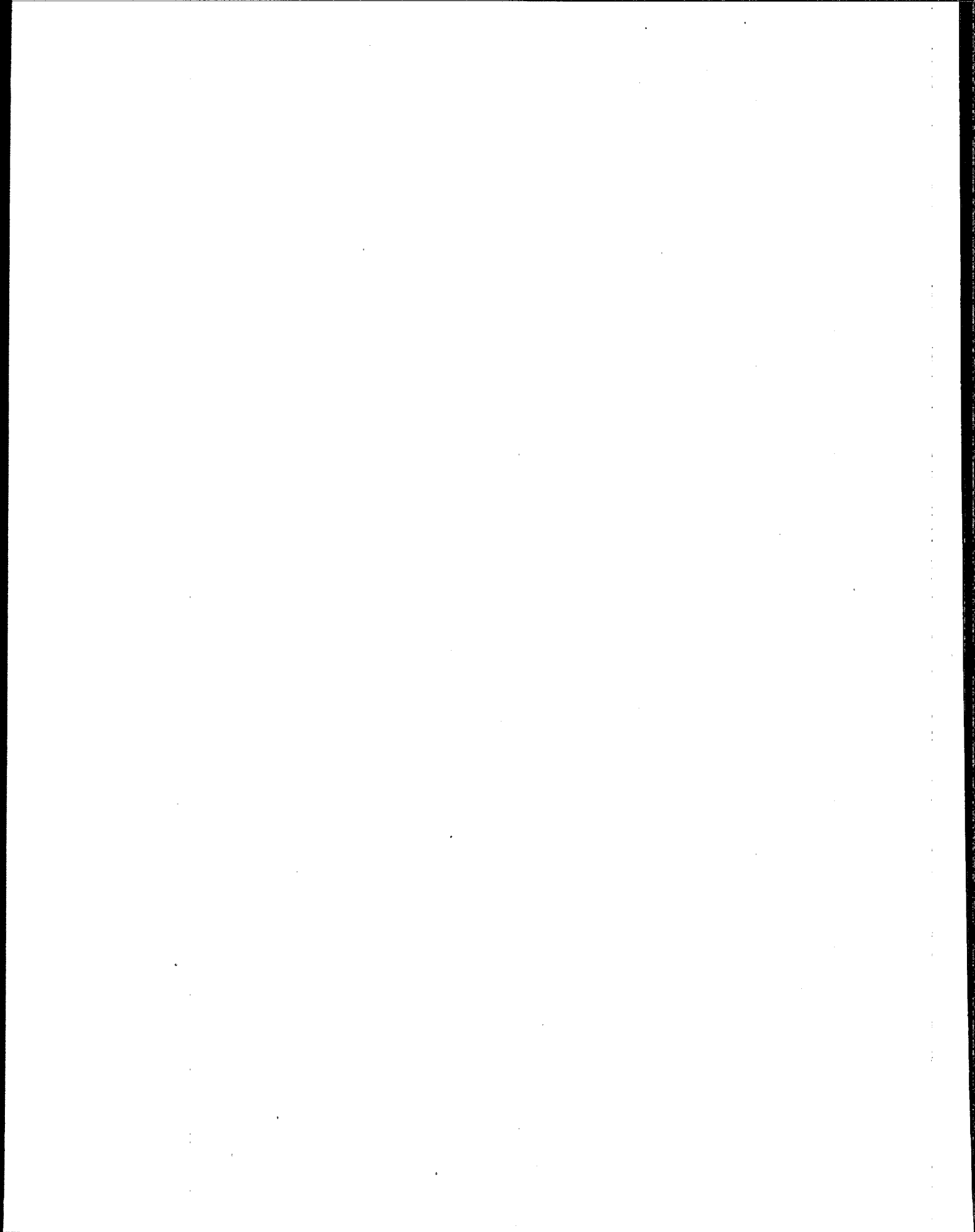


Bi-modally Distributed Windroses



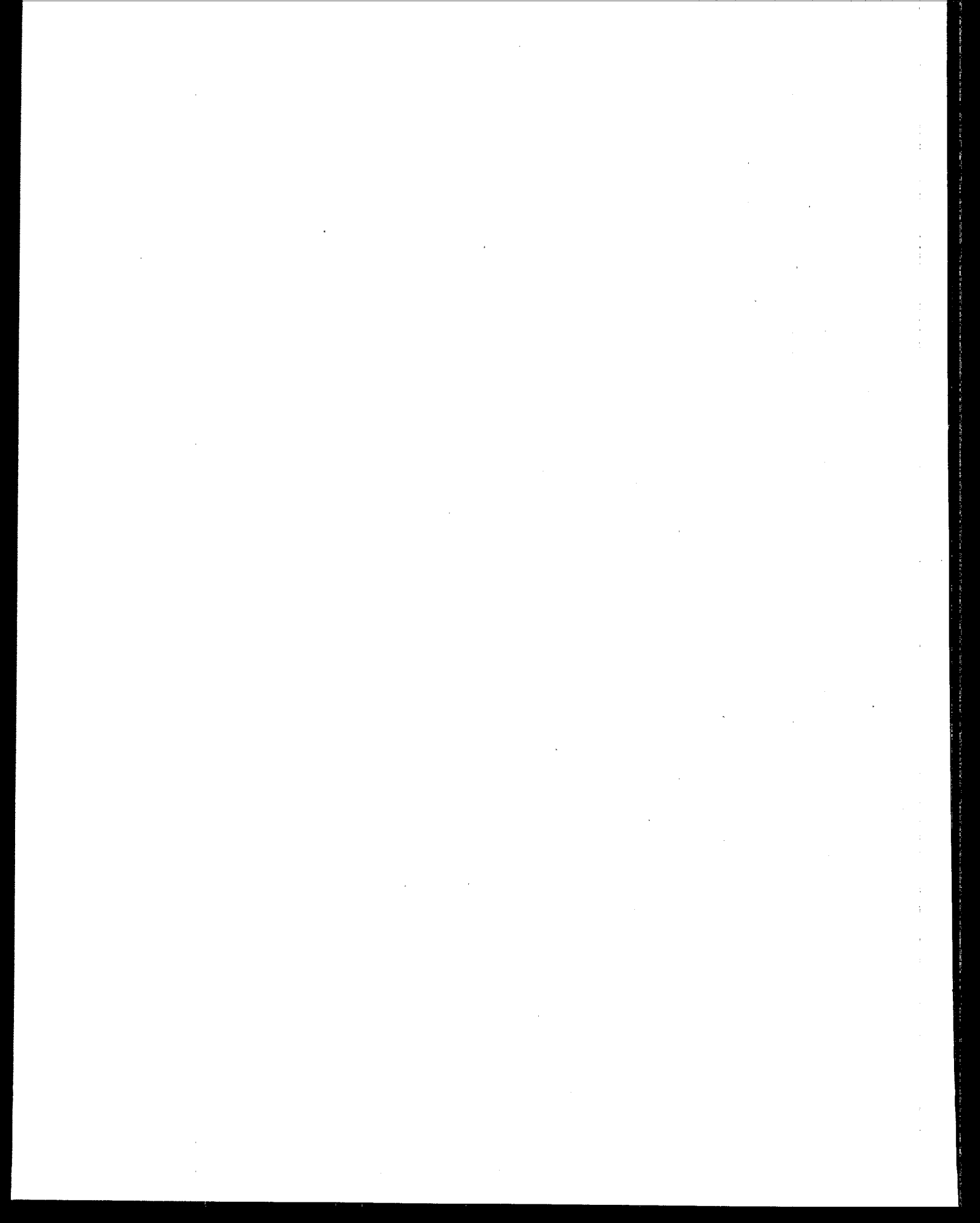
Appendix E

Derivation of Chronic Inhalation Noncancer and Cancer Health Benchmark Values



NONCARCINOGENS

DERIVATION OF INHALATION REFERENCE CONCENTRATIONS



2-Chlorophenol
CAS #95-57-8

RfC:	0.0014 mg/m ³
Basis for RfC:	Route-to-route extrapolation from the RfD
Critical Study:	Exon, J.H., and L.D. Koller. 1982. Effects of transplacental exposure to chlorinated phenols. <i>Environ Health Perspect</i> 46:137-140 (as cited in U.S. EPA, 1998).
Critical Dose:	5 mg/kg/d [X] NOAEL [] LOAEL
Critical Effect:	Increase in conception rate and number of stillbirths and decrease in size of litters
Species:	Rat
Route of Exposure:	Drinking water
Duration:	10 weeks
Uncertainty Factor:	1000: 10 for extrapolation from animals to humans 10 for protection of sensitive human subpopulations 10 for use of a subchronic study
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{RfD} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 0.005 \text{ mg/kg/d} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 0.0014 \text{ mg/m}^3$$

where:

70 kg = default adult human body weight

20 m³/d = default human daily rate of inhalation

Calculations assume 100% absorption.

Summary of Study:

The RfD is based on a NOAEL of 5 mg/kg/d with a LOAEL of 50 mg/kg/d for reproductive effects in a subchronic drinking water study in rats (Exon and Koller, 1982, as cited in U.S. EPA, 1998). In this study, groups of 12 to 20 weanling female Sprague-Dawley rats were exposed to 0, 5, 50, or 500 ppm of 2-chlorophenol in the drinking water and bred after 10 weeks of 2-chlorophenol treatment. Treatment was continued during breeding, gestation, and weaning. The weanling rats were evaluated for percent conception, litter size, birth weight, weaning weight, number of stillbirths, and hematology (hematocrit, hemoglobin levels, red and white cell counts, and mean corpuscular volume). The evaluations revealed an increase in the conception rate and in the number of stillborns as well as a decrease in the size of the litters in the rats

exposed to 500 ppm, which can be converted to a dosage of 50 mg/kg/d—the LOAEL. No effects were observed at 50 ppm, which can be converted to a dosage of 5 mg/kg/d. Dividing the NOAEL of 5 mg/kg/d by an uncertainty factor of 1,000 (10 factors each for animal to human extrapolation, interspecies variability, and the use of subchronic data), yields the RfD of 0.005 mg/kg/d (EPA, 1998).

Rationale for Route-to-Route Extrapolation:

A first pass in the liver or respiratory tract is not expected to contribute to the toxicity of 2-chlorophenol because it has been demonstrated that the toxic action of the lower chlorinated phenols is due to the undissociated molecule. In studies with rats, it was observed that the toxicity of chlorophenols administered via subcutaneous and intraperitoneal routes is similar to that which is observed in orally administered chlorophenols (Deichmann and Keplinger, 1981). Since the dermal irritation index for 2-chlorophenol is low, no significant portal of entry effect is expected from inhalation exposure to 2-chlorophenol (HSDB, 1998).

Consequently, route-specific difference in toxicity is not expected for 2-chlorophenol. Therefore, in accordance with EPA guidelines (U.S. EPA, 1994), the oral toxicity data for 2-chlorophenol are adequate for use in the calculation of an inhalation RfC for the substance.

Strengths and Uncertainties:

The strength of the RfC is that it is based on an RfD on IRIS that has undergone rigorous EPA peer review.

The major uncertainty of the RfC is the lack of inhalation toxicity studies in humans or animals and the use of default values in the route-to-route extrapolation.

References:

- Deichmann, W.B., and M.L. Keplinger. 1981. Aromatic Hydrocarbons. In: G.D. Clayton and F.E. Clayton (eds). *Patty's Industrial Hygiene and Toxicology*. 3rd revised edition. Volume 2A: Toxicology. New York: John Wiley and Sons, pp. 3325-3415.
- Exon, J.H., and L.D. Koller. 1982. Effects of transplacental exposure to chlorinated phenols. *Environ Health Perspect* 46:137-140 (as cited in U.S. EPA, 1998).
- Hazardous Substances Databank (HSDB): 2-Chlorophenol. 1998. Online database. National Library of Medicine, Bethesda, MD.
- U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.
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Cobalt
CAS #7440-48-4

RfC:	0.00001 mg/m ³
Critical Study:	NTP. 1996. NTP Technical Report on the toxicology and carcinogenesis studies of cobalt sulfate heptahydrate (CAS No.10026-24-1) in F344/N rats and B6C3F ₁ mice (inhalation studies). Draft. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH Publication no. 96-3961. NTP TR-471.
Critical Dose:	0.11 mg/m ³ [] NOAEL [X] LOAEL
Critical Effect:	Hyperplasia of the lateral wall of the nose and atrophy of the olfactory epithelium
Species:	Rat
Route of Exposure:	Inhalation
Duration:	104 weeks
Uncertainty Factor:	300: 10 for use of a LOAEL 10 for protection of sensitive human subpopulations 3 for extrapolation from animals to humans with the use of a LOAEL adjusted for human equivalent concentration
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{LOAEL}_{\text{HEC}} \div \text{UF} = 0.004 \text{ mg/m}^3 \div 300 = 0.00001 \text{ mg/m}^3$$

Summary of Study:

Groups of 50 male and 50 female rats and mice were exposed to aerosols containing 0, 0.3, 1.0, and 3.0 mg/m³ cobalt sulfate heptahydrate (0, 0.11, 0.4, and 1.1 mg cobalt/m³) 6 hours/day, 5 days per week, for 104 weeks (NTP, 1996). Survival of both rats and mice were similar to controls. The respiratory tract was the primary site of nonneoplastic lesions and neoplasms. Cobalt sulfate heptahydrate caused a spectrum of inflammatory, fibrotic, and proliferative lesions in the respiratory tract. Olfactory epithelial atrophy and hyperplasia of the lateral wall of the nose were increased significantly at all dose levels in both male and female rats, and severity of these lesions increased with increasing exposure concentration. Olfactory epithelial atrophy was seen at 0.4 and 1.1 mg cobalt/m³ in male and female mice. Hyperplasia of the adrenal medulla was increased significantly in male rats at 0.11 mg cobalt/m³ and cytoplasmic vacuolization of the bronchus was increased significantly in male and female mice at this concentration.

A LOAEL of 0.11 mg cobalt/m³ was selected based on respiratory effects (olfactory epithelial atrophy and hyperplasia of the lateral wall of the nose) observed at this lowest dose in rats. This LOAEL was adjusted for continuous exposure (0.02 mg/m³). A LOAEL_{HEC} was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-5 (insoluble, approximately spherical particles). An uncertainty factor of 300 was applied: 10 for use of a LOAEL, 10 for protection of sensitive human subpopulations, and 3 for extrapolation from animals to humans with the use of a LOAEL adjusted for human equivalent concentration.

Conversion Factors:

dose levels are:

% cobalt in cobalt sulfate heptahydrate = 38% = 0.38.

0.3 mg CoSO₄•7H₂O/m³ x 0.38 = 0.11 mg Co/m³; 1.0 mg CoSO₄•7H₂O/m³ = 0.4 mg Co/m³;

3.0 mg CoSO₄•7H₂O/m³ = 1.1 mg Co/m³.

LOAEL_{ADJ} = 0.11 mg/m³ x (6 hr/24 hr) x (5/7 d) = 0.02 mg/m³

LOAEL_{HEC} = LOAEL_{ADJ} x RDDR_r

LOAEL_{HEC} = 0.02 mg/m³ x 0.209 = 0.004 mg/m³

where

LOAEL_{ADJ} is the adjusted LOAEL, RDDR_r is a multiplicative factor used to adjust an observed inhalation particulate concentration of an animal to the predicted inhalation particulate exposure concentration for a human; based on MMAD = 1.5 μm, sigma g = 2.2, mean body weight of female rat = 237 g, extrathoracic respiratory effects; RDDR calculated using EPA RDDR Program (computer disk).

Additional Information:

Human studies support the critical endpoint (the respiratory system) identified in the NTP (1996) study. Respiratory irritation, wheezing, asthma, pneumonia, and fibrosis have been reported in epidemiological studies at concentrations ranging from 0.007 to 0.893 mg cobalt/m³ (Antilla et al., 1986; Davison et al., 1983; Demedts et al., 1984; Hartung et al., 1982; Shirakawa et al., 1988, 1989; Van Cutsem et al., 1987, as cited in ATSDR, 1992). Sprince et al. (1988) reported a strong relationship between work-related wheezing and cobalt exposure in workers engaged in tungsten carbide production. The relative odds for work-related wheezing doubled when cobalt exposure exceeded 0.050 mg/m³ compared with exposure to less than 0.050 mg/m³.

Animal studies have also shown a variety of respiratory effects. Exposure of rats and rabbits to mixed cobalt oxides resulted in lesions of the alveolar region of the respiratory tract (Johanssen et al., 1984, 1987, as cited in ATSDR, 1992). Lifetime exposure of hamsters to cobalt oxide resulted in emphysema (Wehner et al., 1977, as cited in ATSDR, 1992). An NTP subchronic (13-week) study reported squamous metaplasia of the larynx, histiocytic infiltrates in the lung, and acute inflammation of the nose in rats and mice (NTP, 1991). A LOAEL of 0.3 mg cobalt sulfate heptahydrate/m³ was identified for this endpoint.

Strengths and Uncertainties:

The strengths of the RfC are that it was based on a well-designed chronic study from the NTP that involved extensive clinical and pathological examinations in two species and the critical effect noted in the study has been observed in numerous other human and animal studies.

The major uncertainty of the RfC is the lack of a NOAEL from this study or other studies. The high incidence of adverse effects at the lowest dose tested presents uncertainties as to the true NOAEL for cobalt.

References:

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological profile for cobalt. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Anttila, S., S. Sutinen, M. Paananen, et al. 1986. Hard metal disease: A clinical, histological, ultrastructural and X-ray microanalytical study. *Eur J Respir Dis* 69:83-94 (as cited in ATSDR, 1992).

California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic reference exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.

Davison, A.G., P.L. Haslam, B. Corrin, et al. 1983. Interstitial lung disease and asthma in hard-metal workers: Bronchoalveolar lavage, ultrastructural and analytical findings and results of bronchial provocation tests. *Thorax* 38:119-128 (as cited in ATSDR, 1992).

Demedts, M., B. Gheysens, J. Nagels, et al. 1984. Cobalt lung in diamond polishers. *Am Rev Respir Dis* 130:130-135 (as cited in ATSDR, 1992).

Hartung, M., K.H. Schaller, and E. Brand. 1982. On the question of the pathogenetic importance of cobalt for hard metal fibrosis of the lung. *Int Arch Occup Env Health* 50:53-57 (as cited in ATSDR, 1992).

Johanssen, A., B. Robertson, and P. Camner. 1987. Nodular accumulation of type II cells and inflammatory lesions caused by inhalation of low cobalt concentrations. *Environ Res* 43:227-243 (as cited in ATSDR, 1992).

Johanssen, A., P. Camner, C. Jarstrand, et al. 1984. Lung morphology and phospholipids after experimental inhalation of soluble cadmium, copper, and cobalt. *Environ Res* 34:295-309 (as cited in ATSDR, 1992).

NTP. 1991. NTP report on the toxicity studies of cobalt sulfate heptahydrate in F344/N rats and B6C3F₁ mice (inhalation studies). Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH Publication no. 91-3124. NTP TOX-5.

NTP. 1996. NTP Technical Report on the toxicology and carcinogenesis studies of cobalt sulfate heptahydrate (CAS No.10026-24-1) in F344/N rats and B6C3F₁ mice (inhalation studies). Draft. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health. NIH Publication no. 96-3961. NTP TR-471.

Shirakawa, T., Y. Kusaka, N. Fujimura, et al. 1988. The existence of specific antibodies to cobalt in hard metal asthma. *Clin Allergy* 18:451-460 (as cited in ATSDR, 1992).

Shirakawa, T., Y. Kusaka, N. Fujimura, et al. 1989. Occupational asthma from cobalt sensitivity in workers exposed to hard metal dust. *Chest* 95:29-37 (as cited in ATSDR, 1992).

Sprince, N.L., L.C. Oliver, E.A. Eisen, R.E. Greene, and R.I. Chamberlin. 1988. Cobalt exposure and lung disease in tungsten carbide production: a cross-sectional study of current workers. *Am Rev Respir Dis* 138:1220-1226.

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Van Cutsem, E.J., J.L. Ceuppens, L.M. Lacquet, et al. 1987. Combined asthma and alveolitis induced by cobalt in a diamond polisher. *Eur J Respir Dis* 70:54-61 (as cited in ATSDR, 1992).

Wehner, A.P., R.H. Busch, R.J. Olson, et al. 1977. Chronic inhalation of cobalt oxide and cigarette smoke by hamsters. *Am Ind Hyg Assoc J* 38:338-346 (as cited in ATSDR, 1992).

Cresols
CAS #1319-77-3

RfC:	0.0004 mg/m ³
Critical Study:	Uzhdavini, E.R., K. Astaf'yeva, A.A. Mamayeva, and G.Z. Bakhtizina. 1972. [Inhalation toxicity of o-cresol]. Trudy Ufimskogo Nauchno-Issledovatel'skogo Instituta Gigiyeny Profzabolevaniya, 7:115-9. (Russian) [as cited in CalEPA, 1997, and U.S. EPA 1985, 1986]
Critical Dose:	9 mg/m ³ [] NOAEL [X] LOAEL
Critical Effect:	Alterations in bone marrow cellularity
Species:	Rat
Route of Exposure:	Inhalation
Duration:	4 months
Uncertainty Factor:	3000: 10 for use of a LOAEL 10 for extrapolation from animals to humans 10 for protection of sensitive human subpopulations 3 for extrapolation from subchronic to chronic exposure
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{LOAEL}_{\text{HEC}} \div \text{UF} = 1.3 \text{ mg/m}^3 \div 3000 = 0.0004 \text{ mg/m}^3$$

Summary of Study:

Male and female rats were exposed to 0 or 9.0 mg/m³ o-cresol via inhalation, first for 2 months (6 h/d, 5 d/wk) and then for 2 more months (4 h/d, 5 d/wk) (Uzhdavini et al., 1972, as cited in CalEPA, 1997). The following endpoints were examined: elemental conditioned defensive reflex, white blood cell levels, bone marrow elements, and liver function (as indicated indirectly by hexobarbital narcosis). Both exposed and control animals showed some loss of the defensive reflex, with the effect occurring in all exposed animals before the end of the second month and in control animals at later times. White blood cell counts were elevated in male animals, peaking at the end of the exposure period and returning to normal 1 month after cessation of exposure. Exposed animals also showed a statistically significant change in the leukoid-to-erythroid ratio in the bone marrow. Liver toxicity was suggested by an extension of hexobarbital narcosis duration in treated animals. A LOAEL of 9 mg/m³ for hematological effects was identified.

The LOAEL of 9 mg/m³ was adjusted for continuous exposure (1.3 mg/m³). A LOAEL_{HEC} was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrarrespiratory effects). An uncertainty factor of 3000 was applied: 10 for use of a

LOAEL, 10 for extrapolation from humans to animals, 10 for human variability, and 3 for extrapolation from subchronic to chronic exposure.

Conversion Factors:

$$\text{LOAEL}_{\text{ADJ}} = 9 \text{ mg/m}^3 \times (5/24 \text{ h}) \times (5/7 \text{ d}) = 1.3 \text{ mg/m}^3$$

$$\text{LOAEL}_{\text{HEC}} = \text{LOAEL}_{\text{ADJ}} \times \text{RGDR}$$

$$\text{LOAEL}_{\text{HEC}} = \text{LOAEL}_{\text{ADJ}} \times (\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$$

$$\text{LOAEL}_{\text{HEC}} = 1.3 \text{ mg/m}^3 \times 1 = 1.3 \text{ mg/m}^3$$

where

$\text{LOAEL}_{\text{ADJ}}$ is the adjusted LOAEL, RGDR is the regional gas dose ratio (animal:human), and $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ is the ratio of blood:gas partition coefficient; $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ defaults to 1 where $\text{H}_{\text{b/g}}$ values are not known.

Additional Information:

In humans, inhalation exposure is reported to cause respiratory effects, including the development of pneumonia, pulmonary edema, and hemorrhage (Clayton and Clayton, 1981). Irritation of the nose and throat, nasal constriction, and dryness was reported in 8 of 10 individuals briefly exposed to 6 mg/m³ (Uzhdavini et al., 1972, as cited in CalEPA 1997).

Signs of respiratory irritation (as indicated by increased paratid gland secretions) were observed in cats exposed to 5 to 9 mg/m³ o-cresol for 30 minutes (Uzhdavini et al., 1972, as cited in CalEPA 1997). Exposure of mice to 50 mg/m³ o-cresol for 2 h/d for 1 month did not affect mortality; however, heart muscle degeneration and degeneration of nerve cells and glial elements were reported (Uzhdavini et al., 1972, as cited in CalEPA, 1997, U.S. EPA, 1985).

Strengths and Uncertainties:

Major areas of uncertainty are the lack of human data, the scarcity of animal inhalation data, and the lack of a NOAEL for this study. Also, the data presented were incomplete, the number of animals used is not known, exposure and control conditions were not described, statistical analyses were not provided, and the purity of the compound tested could not be ascertained.

References:

California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic reference exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.

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U.S. Environmental Protection Agency. 1985. Health and environmental effects profile for cresols. Cincinnati, OH: Environmental Criteria and Assessment Office, Office of Research and Development, U.S. EPA. EPA/600/x-85-358.

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U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Uzhdavini, E.R., K. Astaf'yeva, A.A. Mamayeva, and G.Z. Bakhtizina. 1972. [Inhalation toxicity of o-cresol]. Trudy Ufimskogo Nauchno-Issledovatel'skogo Instituta Gigiyeny Profzabolevaniya, 7:115-9. [as cited in CalEPA, 1997, and U.S. EPA 1985, 1986]

3,4-Dimethylphenol CAS # 95-65-8

RfC: Data are inadequate to support the derivation of an RfC at this time.

Supporting Data:

An RfD of 0.001 mg/kg/d is listed in IRIS (U.S. EPA, 1998), based on a subchronic feeding study in rats. Changes in blood pressure and body weight and histopathological changes in liver, kidney and spleen were reported (Veldre and Janes, 1979). Route-to-route extrapolation of an RfC from the RfD is not recommended because of the potential for respiratory tract effects following inhalation exposure and first-pass effects following ingestion exposure.

Although dimethylphenols have been detected in tobacco smoke, automobile exhausts, and exhausts from stationary sources, they have not been detected in ambient air (U.S. EPA, 1986). 3,4-Dimethylphenol is not likely to occur at detectable concentrations in ambient air because it is a solid at ambient temperatures and has a low vapor pressure. Consequently, inhalation exposures are unlikely to be important for the general population. Skin absorption and ingestion, which can be evaluated by the RfD, are likely to be the predominant exposure pathways.

Very little toxicity or metabolism data specific to 3,4-dimethylphenol are available. Dimethylphenols and related compounds (phenol and methylphenols [cresols]) are rapidly absorbed following ingestion, inhalation, or skin contact and are corrosive to skin, eyes, mucous membranes, and the respiratory tract. Therefore, portal-of-entry effects are likely to be important and cannot be addressed from route-to-route extrapolation. First-pass effects also may be important. These compounds are metabolized predominantly to glucuronide and sulfate conjugates and excreted in the urine (U.S. EPA, 1986). Skowronski et al. (1994) suggested that a lack of first-pass metabolism in the liver may contribute to the toxicity of phenol following skin absorption; therefore, differences in metabolism following ingestion and inhalation exposures also could affect toxicity.

References:

Skowronski, G.A., A.M. Kadry, R.M. Turkall, et al. 1994. Soil decreases the dermal penetration of phenol in male pig in vitro. *J Toxicol Environ Health* 41:467-479.

U.S. Environmental Protection Agency. 1986. Health and environmental effects profile for dimethylphenols. Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/x-86/256.

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA /600/8-90-066F.

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Veldre, I.A., and H.J. Janes. 1979. Toxicological studies of shale oils, some of their components and commercial products. *Environ Health Perspect* 30:141-146 (as cited in U.S. EPA, 1998).

1,4-Dioxane
CAS # 123-91-1

RfC:	0.8 mg/m ³
Critical Study:	Torkelson, T.R., B.K.J. Leong, R.J. Kociba, et al. 1974. 1,4-Dioxane. II. Results of a 2-year inhalation study in rats. <i>Toxicol Appl Pharmacol</i> 30:287-298.
Critical Dose:	400 mg/m ³ [X] NOAEL [] LOAEL
Critical Effect:	No effect on liver, kidney, or hematological endpoints
Species:	Rat
Route of Exposure:	Inhalation
Duration:	2 years
Uncertainty Factor:	100: 10 for extrapolation from animals to humans 10 for protection of sensitive human subpopulations
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{NOAEL}_{\text{HEC}} \div \text{UF} = 83.3 \text{ mg/m}^3 \div 100 = 0.8 \text{ mg/m}^3 (0.2 \text{ ppm})$$

Summary of Study:

Groups of Wistar rats were exposed to 0 or 111 ppm (0 or 400 mg/m³) 1,4-dioxane 7 h/d, 5 d/wk for 2 years (Torkelson et al., 1974). Animals were observed for signs of toxicity, including behavioral changes, eye and nasal irritation, respiratory distress, and skin condition. Body weight was measured weekly. Hematological measurements were made at 16 and 23 months and included serum glutamic-pyruvic transaminase (SGPT) activity, blood urea nitrogen (BUN), alkaline phosphatase (AP) activity, and total protein determinations. At sacrifice, gross necropsy of all animals was performed, and organs were examined for tumors. Histological examination of tissues was conducted.

No significant differences in survival, body weight, general appearance, or behavior were reported. Packed cell volume (PCV), red blood cells, and hemoglobin were slightly, but significantly ($p < 0.05$), increased and white blood cells were significantly decreased in exposed males; however, the study authors note that these differences were within normal physiological levels and not considered of toxic importance. Slightly decreased BUN and AP values observed in exposed males were not considered to be biologically significant by the investigators based on the fact that an increase, not a decrease, in these parameters would indicate kidney or liver damage. Increased total protein in exposed males was also reported but not considered to be biologically significant. No significant differences in liver, kidney, or spleen weights, or gross or

microscopic alterations were observed. Tumor incidence (including hepatic and nasal) was not significantly different in any of the organs examined.

The NOAEL of 400 mg/m³ was adjusted for continuous exposure (83.3 mg/m³). A NOAEL_{HEC} was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrapulmonary effects). An uncertainty factor of 100 was applied: 10 for extrapolation from humans to animals and 10 for human variability.

Conversion Factors:

$$0.4 \text{ mg/L} \times 1,000 \text{ L/m}^3 = 400 \text{ mg/m}^3$$

$$\text{NOAEL}_{\text{ADJ}} = 400 \text{ mg/m}^3 \times (7/24 \text{ hr}) \times (5/7 \text{ d}) = 83.3 \text{ mg/m}^3$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times \text{RGDR}$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times (H_{\text{b/g}})_{\text{A}} / (H_{\text{b/g}})_{\text{H}}$$

$$\text{NOAEL}_{\text{HEC}} = 83.3 \text{ mg/m}^3 \times 1 = 83.3 \text{ mg/m}^3$$

where

NOAEL_{ADJ} is the adjusted NOAEL, RGDR is the regional gas dose ratio (animal:human), and $(H_{\text{b/g}})_{\text{A}} / (H_{\text{b/g}})_{\text{H}}$ is the ratio of blood:gas partition coefficient; $(H_{\text{b/g}})_{\text{A}} / (H_{\text{b/g}})_{\text{H}}$ defaults to 1 where $H_{\text{b/g}}$ values are not known.

Additional Information:

The major metabolite of 1,4-dioxane in rats is beta-hydroxyethoxyacetic acid (HEAA), which is excreted in the urine (Braun and Young, 1977). Results from a study by Young et al. (1978) show that the fate of 1,4-dioxane in rats is markedly dose-dependent due to a limited capacity to metabolize dioxane to HEAA. Exposure to 1,4-dioxane by ingestion results in saturation of metabolism above a single dose of 100 mg/kg, or as low as 10 mg/kg when administered in multiple doses. When rats were exposed to 50 ppm for 6 hours, nearly all the inhaled 1,4-dioxane was also metabolized to HEAA (99%); the plasma half-life was 1.1 hours (Young et al., 1978). The correlation of the dose-dependent fate of 1,4-dioxane with the results of toxicological studies in rats supports the conclusion that there is an apparent threshold for the toxic effects of dioxane that coincides with saturation of the metabolic pathway for its detoxification (Young et al., 1978). 1,4-Dioxane and HEAA were also found in the urine of dioxane plant workers exposed to an average concentration of 1.6 ppm (TWA) for 7.5 hours (Young et al., 1976, 1977).

In a study by Kociba et al. (1974), Sherman rats were exposed to 0, 0.01, 0.1, or 1.0% 1,4-dioxane in drinking water for up to 2 years. No hematologic changes were reported. Histopathologic examination revealed hepatocellular and renal tubular degenerative changes, accompanied by regenerative activity, in rats exposed to the two highest dose levels, but not at the low dose (Kociba et al., 1974). The lack of hematological effects observed in the ingestion study suggests that the toxicity of 1,4-dioxane may be route-specific. Studies suggest that the inhalation of 1,4-dioxane may lead to adverse effects, but good dose-response data are not available. The toxicity of 1,4-dioxane may be a function of the saturation of the mechanism of metabolism (Young et al., 1978).

Strengths and Uncertainties:

The strengths of the RfC are that it is based on a lifetime study, with a large number of toxic endpoints examined and a large sample size (n=192-288). The weaknesses of the inhalation benchmark value include the use of a free-standing NOAEL, that only one exposure level was used in the Torkelson et al. (1974) study, the limited human data, the limited inhalation data in animals, and the lack of developmental and reproductive studies.

References:

Braun, W.H., and J.D. Young. 1977. Identification of beta-hydroxyethoxyacetic acid the major urinary metabolite of 1,4-dioxane in the rat. *Toxicol Appl Pharmacol* 39:33-38.

California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic reference exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.

Kociba, R.J., S.B. McCollister, C. Park, et al. 1974. 1,4-Dioxane. I. Results of a 2-year ingestion study in rats. *Toxicol Appl Pharmacol* 30:275-286.

Torkelson, T.R., B.K.J. Leong, R.J. Kociba, et al. 1974. 1,4-Dioxane. II. Results of a 2-year inhalation study in rats. *Toxicol Appl Pharmacol* 30:287-298.

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Young, J.D., W.H. Braun, and P.J. Gehring. 1978. Dose-dependent fate of 1,4-dioxane in rats. *J Toxicol Environ Health* 4:709-726.

Young, J.D., W.H. Braun, P.J. Gehring, et al. 1976. 1,4-Dioxane and beta-hydroxyethoxyacetic acid excretion in urine of humans exposed to dioxane vapors. *Toxicol Appl Pharmacol* 38:643-646.

Young, J.D., W.H. Braun, L.W. Rampy, et al. 1977. Pharmacokinetics of 1,4-dioxane in humans. *J Toxicol Environ Health* 3:507-520.

2-Ethoxyethanol Acetate
CAS # 111-15-9

RfC:	0.3 mg/m ³
Basis for RfC:	Calculated from RfC for 2-ethoxyethanol
Critical Study:	Barbee, S.J., J.B. Terrill, D.J. DeSousa, and C.C. Conaway. 1984. Subchronic inhalation toxicology of ethylene glycol monoethyl ether in the rat and rabbit. <i>Environ Health Perspect</i> 57: 157-163.
Critical Dose:	68 mg/m ³ (2-ethoxyethanol) [X] NOAEL [] LOAEL
Critical Effect:	Decreased testis weight, seminiferous tubule degeneration, and decreased hemoglobin (2-ethoxyethanol)
Species:	Rabbit
Route of Exposure:	Inhalation
Duration:	13 weeks
Uncertainty Factor:	300: 3 for extrapolation from animals to humans 10 for intraspecies extrapolation 10 for use of a subchronic study
Modifying Factor:	1

Summary of Study:

Groups of New Zealand rabbits (10/sex/group) were exposed to 0, 25, 103 or 403 ppm (0, 92, 380, or 1,485 mg/m³) 2-ethoxyethanol 6 h/d, 5 d/wk for 13 weeks (Barbee et al., 1984). The duration-adjusted exposure concentrations were 0, 16, 68, and 265 mg/m³, respectively. Groups of Sprague-Dawley rats were similarly exposed to 2-ethoxyethanol (0, 25, 103, or 403 ppm). Physical examination and body weight measurements were conducted weekly. Hematological, chemical, and histopathological changes (including bone marrow of the sternum) were also assessed. No respiratory effects (nasal turbinates, tracheas, and lungs were examined) were observed in either species.

In both male and female rabbits, body weight was significantly depressed at 403 ppm. Significantly decreased testes weight was observed in rabbits exposed to 403 ppm. Pathological changes in the testes were characterized as minimal to slight focal degeneration of the seminiferous tubules with loss of epithelium in 3 of 10 rabbits. Spermatogenic activity in the affected males was judged by overall organ morphology and deemed normal. Additionally, both sexes exhibited significantly decreased hemoglobin, hematocrit, and erythrocyte count at 403 ppm.

Hematological, reproductive, and developmental effects were also observed in pregnant Wistar rats (Doe, 1984). Neurobehavioral and neurochemical effects were reported in the offspring of female Sprague-Dawley rats, as well as in the dams (Nelson et al., 1981, 1982a, 1982b).

Based on the observed testicular and hematopoietic effects at 403 ppm (Barbee et al., 1984), the $\text{NOAEL}_{\text{HEC}}$ and $\text{LOAEL}_{\text{HEC}}$ for 2-ethoxyethanol in the rabbit are identified as 68 and 265 mg/m^3 , respectively. The NOAEL was adjusted for intermittent exposure. A $\text{NOAEL}_{\text{HEC}}$ was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrapulmonary effects). An uncertainty factor of 300 was applied (10 for intraspecies extrapolation, 10 for use of a subchronic study, and 3 to account for interspecies extrapolation). An RfC of 0.2 mg/m^3 was calculated for 2-ethoxyethanol (U.S. EPA, 1998).

Calculations and Conversion Factors:

Dose levels are:

25 ppm 2-ethoxyethanol $\times (90.12/24.45) = 92 \text{ mg}/\text{m}^3$ 2-ethoxyethanol; 103 ppm = 380 mg/m^3 ;
403 ppm = 1,485 mg/m^3 .

$$\text{NOAEL}_{\text{ADJ}} = 380 \text{ mg}/\text{m}^3 \times (6/24 \text{ h}) \times (5/7 \text{ d}) = 68 \text{ mg}/\text{m}^3$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times \text{RGDR}$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times (\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$$

$$\text{NOAEL}_{\text{HEC}} = 68 \text{ mg}/\text{m}^3 \times 1 = 68 \text{ mg}/\text{m}^3$$

where

$\text{NOAEL}_{\text{ADJ}}$ is the adjusted NOAEL , RGDR is the regional gas dose ratio (animal:human), and $(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$ is the ratio of blood:gas partition coefficients; $(\text{H}_{\text{b/g}})_{\text{A}}/(\text{H}_{\text{b/g}})_{\text{H}}$ defaults to 1 where $\text{H}_{\text{b/g}}$ values are not known.

$$\text{RfC for 2-ethoxyethanol} = \text{NOAEL}_{\text{HEC}} \div \text{UF} = 68 \text{ mg}/\text{m}^3 \div 300 = 0.2 \text{ mg}/\text{m}^3$$

$$\text{RfC (2-ethoxyethanol)} \times [\text{MW (2-ethoxyethanol acetate)} \div \text{MW (2-ethoxyethanol)}] =$$

$$\text{RfC (2-ethoxyethanol acetate)}$$

$$0.2 \text{ mg}/\text{m}^3 \times (132.16/90.12) = 0.3 \text{ mg}/\text{m}^3 = \text{RfC (2-ethoxyethanol acetate)}$$

Additional Information:

The RfC of 0.2 mg/m^3 for 2-ethoxyethanol (U.S. EPA, 1998) is based on hematological and male reproductive effects in New Zealand rabbits. It is assumed that 2-ethoxyethanol acetate would be equitoxic to 2-ethoxyethanol on a molar basis, since both compounds are metabolized to ethoxyacetic acid by the alcohol dehydrogenase pathway (DOL-OSHA, 1993). The RfC for 2-ethoxyethanol acetate was calculated from the RfC for 2-ethoxyethanol of 0.2 mg/m^3 (U.S. EPA, 1998), resulting in an RfC of 0.3 mg/m^3 for 2-ethoxyethanol acetate.

Strengths and Uncertainties:

The strength of the RfC is that it is based on an RfC in IRIS that has undergone Agency review. The major uncertainty of the RfC for 2-ethoxyethanol acetate is that it is based on an RfC for 2-ethoxyethanol and converted using the molecular weight ratio. However, it is assumed that 2-ethoxyethanol acetate would be equitoxic to 2-ethoxyethanol on a molar basis.

References:

Barbee, S.J., J.B. Terrill, D.J. DeSousa, and C.C. Conaway. 1984. Subchronic inhalation toxicology of ethylene glycol monoethyl ether in the rat and rabbit. *Environ Health Perspect* 57: 157-163.

Doe, J.E. 1984. Ethylene glycol monoethyl ether and ethylene glycol monoethyl ether acetate teratology studies. *Environ Health Perspect* 57:33-41.

Department of Labor, Occupational Safety and Health Administration (DOL-OSHA). 1993. Federal Register: Occupational exposure to 2-methoxyethanol, 2-ethoxyethanol and their acetates (glycol ethers); Proposed rule. 58FR15526. March 23, 1993.

Nelson, B.K., W.S. Brightwell, J.V. Setzer, et al. 1981. Ethoxyethanol behavioral teratology in rats. *Neurotoxicology* 2:231-249.

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Nelson, B.K., W.S. Brightwell, J.V. Setzer, and T.L. O'Donohue. 1982b. Prenatal interactions between ethanol and the industrial solvent 2-ethoxyethanol in rats: Neurochemical effects in the offspring. *Neurobehav Toxicol Teratol* 4(3):395-401.

Research Triangle Institute (RTI). 1996. Assessment of Risks from the Management of Used Solvents. U.S. Environmental Protection Agency, Office of Solid Waste, Washington, DC. July 1996. [In support of 61FR42317]

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

U.S. Environmental Protection Agency. 1996. Federal Register: Hazardous Waste Management System; Identification and Listing of Hazardous Waste; Solvents; CERCLA Hazardous Substance Designation and Reportable Quantities; Proposed Rule. 61FR42317-354. August 14, 1996.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS) file for 2-ethoxyethanol.

Ethylene glycol
CAS # 107-21-1

RfC:	0.6 mg/m ³
Critical Study:	Wills, J.H., F. Coulston, E.S. Harris, et al. 1974. Inhalation of aerosolized ethylene glycol by man. <i>Clin Toxicol</i> 7:463-476.
Critical Dose:	67 mg/m ³ [X] NOAEL [] LOAEL
Critical Effect:	Throat and upper respiratory tract irritation
Species:	Humans
Route of Exposure:	Inhalation
Duration:	30 days
Uncertainty Factor:	100: 10 for protection of sensitive human subpopulations 10 for use of a subchronic study
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{NOAEL}_{\text{ADJ}} \div \text{UF} = 55.8 \text{ mg/m}^3 \div 100 = 0.6 \text{ mg/m}^3$$

Summary of Study:

Twenty volunteer male prisoners were exposed to ethylene glycol in mean daily concentrations between 3 and 67 mg/m³ for 30 days, 20 h/d, without effect (Wills et al., 1974). Irritation was noted after 15 minutes at an exposure concentration of 188 mg/m³ and was judged intolerable at 244 mg/m³. No effects were observed in clinical serum enzyme levels for liver and kidney toxicity, hematotoxicity, or psychological responses. The irritation resolved soon after exposure with no effects noted after a 6-week followup period.

A NOAEL of 67 mg/m³ was selected and adjusted for continuous exposure (55.8 mg/m³). An uncertainty factor of 100 was applied: 10 for use of a subchronic study (30 day-duration) and 10 for protection of sensitive human subpopulations.

Conversion Factors:

$$\text{NOAEL}_{\text{ADJ}} = 67 \text{ mg/m}^3 \times 20/24 \text{ h} = 55.8 \text{ mg/m}^3$$

Additional Information:

Animal studies are inconclusive regarding the respiratory effects of ethylene glycol. Suber et al. (1989, as cited in ATSDR, 1997) report thickened respiratory epithelium with enlarged goblet cells in rats that inhaled ethylene glycol over 90 days. Another study in rhesus monkeys and rats showed no respiratory effects from continuous exposure to propylene glycol for 13 to 18 months

(Robertson et al., 1947, as cited in ATSDR, 1997). Developmental effects have been seen in animal studies. Tyl et al. (1995a, 1995b, as cited in CalEPA, 1997) reported reduced ossification in humerus, zygomatic arch, and the metatarsals in fetuses of rats and mice exposed to ethylene glycol on days 6 through 15 of gestation.

Strengths and Uncertainties:

The major strength of the RfC is that it was based on human data with controlled inhalation exposures and the observation of a NOAEL. The major uncertainty to the RfC is the lack of chronic inhalation studies in humans and confirming studies in animals.

References:

Agency for Toxic Substances and Disease Registry. 1997. Toxicological profile for ethylene glycol and propylene glycol. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.

Robertson, O.H., C.G. Loosli, and T.T. Puck. 1947. Test for chronic toxicity of propylene glycol and triethylene glycol on monkeys and rats by vapor inhalation and oral administration. *J. Pharmacol Exper Therap* 91:52-76 (as cited in ATSDR 1997).

Suber, R.L., R.D. Deskin, I. Nikiforov, et al. 1989. Subchronic nose-only inhalation study of propylene glycol in Sprague-Dawley rats. *Food Chem Toxicol* 27(9):573-584 (as cited in ATSDR, 1997).

Tyl, R.W., B. Ballantyne, L.C. Fisher, et al. 1995a. Evaluation of the developmental toxicity of ethylene glycol aerosol in CD-1 mice by nose-only exposure. *Fundam Appl Toxicol* 27:49-62 (as cited in CalEPA, 1997).

Tyl, R.W., B. Ballantyne, L.C. Fisher, et al. 1995b. Evaluation of the developmental toxicity of ethylene glycol aerosol in CD rat and CD-1 mouse by whole-body exposure. *Fundam Appl Toxicol* 24:57-75 (as cited in CalEPA, 1997).

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Wills, J.H., F. Coulston, E.S. Harris, et al. 1974. Inhalation of aerosolized ethylene glycol by man. *Clin Toxicol* 7:463-476.

Methanol
CAS # 67-56-1

RfC:	13 mg/m ³
Critical Study:	Rogers, J.M., M.L. Mole, N. Chernoff, et al. 1993. The developmental toxicity of inhaled methanol in the CD-1 mouse, with quantitative dose-response modeling for estimation of benchmark doses. <i>Teratology</i> 47(3):175-188.
Critical Dose:	1,310 mg/m ³ [X] NOAEL [] LOAEL
Critical Effects:	Developmental malformations (increased cervical ribs, exencephaly, and cleft palate)
Species:	Mouse
Route of Exposure:	Inhalation
Duration:	Gd 6-15
Uncertainty Factor:	100: 10 for extrapolation from animals to humans 10 for protection of sensitive human subpopulations
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{NOAEL}_{\text{HEC}} \div \text{UF} = 1310 \text{ mg/m}^3 \div 100 = 13 \text{ mg/m}^3 (10 \text{ ppm})$$

Summary of Study:

Groups of pregnant CD-1 mice were exposed to 1,000, 2,000, 5,000, 7,500, 10,000, or 15,000 ppm methanol (1,310, 2,620, 6,552, 9,828, 13,104, or 19,656 mg/m³) for 7 h/d on days 6 through 15 of gestation (Rogers et al., 1993). Three groups of controls were used. Sham-exposed controls were exposed to filtered air. Additional control groups remained in their cages and received food and water ad libitum or were food-deprived for 7 h/d (to match the food deprivation experienced by the exposed mice). Dams were observed twice daily and weighed on alternate days during the exposure period. Blood methanol concentrations were determined in three mice per exposure level on gestation days 6, 10, and 15. On day 17, the remaining mice were weighed and sacrificed and the gravid uteri removed. Implantation sites, live and dead fetuses, and resorptions were counted, and fetuses were examined externally and weighed as a litter. Half of each litter were examined for skeletal morphology and the other half of each litter were examined for internal soft tissue anomalies.

One dam died in each of the three highest exposure groups, but no dose-response relationship was evident for maternal death. The sham-exposed and food-deprived controls, as well as all methanol-exposed dams, gained less weight than did unexposed dams fed ad libitum, but

methanol did not exacerbate this effect. Significant increases in the incidence of exencephaly and cleft palate were observed at 6,552 mg/m³ and above, increased embryo/fetal death at 9,828 mg/m³ and above (including an increasing incidence of full-litter resorptions), and reduced fetal weight at 13,104 mg/m³ and above. A dose-related increase in cervical ribs (small ossification sites lateral to the seventh cervical vertebra) was significant at 2,620 mg/m³ and above. Therefore, a NOAEL of 1,310 mg/m³ for developmental toxicity in mice was identified in this study.

Because this is a developmental study, the NOAEL of 1,310 mg/m³ was not adjusted for continuous exposure. A NOAEL_{HEC} was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrarrespiratory effects). An uncertainty factor of 100 was applied: 10 for extrapolation from humans to animals and 10 for human variability.

Conversion Factors:

Dose levels are:

(1,000 ppm x 32.04)/ 24.45 = 1,310 mg/m³; 2,000 ppm = 2,620 mg/m³; 5,000 ppm = 6,552 mg/m³; 7,500 ppm = 9,828 mg/m³; 10,000 ppm = 13,104 mg/m³; 15,000 ppm = 19,656 mg/m³

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL} \times \text{RGDR}$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL} \times (\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$$

$$\text{NOAEL}_{\text{HEC}} = 1310 \text{ mg/m}^3 \times 1 = 1310 \text{ mg/m}^3$$

where

RGDR is the regional gas dose ratio (animal:human) and $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ is the ratio of blood:gas partition coefficient; $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ defaults to 1 where $\text{H}_{\text{b/g}}$ values are not known.

Additional Information:

Developmental effects were also reported in a study by Nelson et al. (1985). Pregnant Sprague-Dawley rats were exposed to methanol at concentrations of 0, 5,000, 10,000, and 20,000 ppm (0, 6,552, 13,104, and 26,208 mg/m³) 7 h/d on days 1 through 19 of gestation (high dose rats were exposed on Gd 7-15 only). Dams were sacrificed on Day 20. Half of the fetuses were examined for visceral defects, and the other half were examined for skeletal defects. No effect on the numbers of corpora lutea or implantations or the percentage of dead or resorbed fetuses was observed. At the two highest concentrations, a dose-related decrease in fetal weights was reported. The highest concentration of methanol produced slight maternal toxicity and a high incidence of congenital malformations ($p < 0.001$), predominantly extra or rudimentary cervical ribs and urinary or cardiovascular defects. Similar malformations were seen in the 10,000 ppm group, but the incidence was not significantly different from controls. No adverse effects were noted in the 6552 mg/m³ group (Nelson et al., 1985).

Strengths and Uncertainties:

The major strengths of the Rogers et al. (1993) study are the identification of a NOAEL and the demonstration of a dose-response relationship. The study was well performed, large numbers of

animals were used (n=20-44 per group), and effects at six exposure concentrations were examined. The results are also supported by an additional developmental study (Nelson et al., 1985).

The major uncertainties of the RfC are the lack of human data for chronic inhalation exposure and the lack of comprehensive, long-term multiple dose studies.

References:

California Environmental Protection Agency (CalEPA). 1997. Technical support document for the determination of noncancer chronic reference exposure levels, Draft for Public Review. Office of Environmental Health Hazard Assessment, Air Toxicology and Epidemiology Section, Berkeley, CA.

Nelson, B.K., W.S. Brightwell, D.R. MacKenzie, et al. 1985. Teratological assessment of methanol and ethanol at high inhalation levels in rats. *Fundam Appl Toxicol* 5:727-736.

Rogers, J.M., M.L. Mole, N. Chernoff, et al. 1993. The developmental toxicity of inhaled methanol in the CD-1 mouse, with quantitative dose-response modeling for estimation of benchmark doses. *Teratology* 47(3):175-188.

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

2-Methoxyethanol Acetate
CAS # 110-49-6

RfC:	0.03 mg/m ³
Basis for RfC:	Calculated from RfC for 2-methoxyethanol
Critical Study:	Miller, R.R., J.A. Ayres, J.T. Young, and M.J. McKenna. 1983. Ethylene glycol monomethyl ether. I. Subchronic vapor inhalation study with rats and rabbits. <i>Fund Appl Toxicol</i> 3(1):49-54.
Critical Dose:	17 mg/m ³ (2-methoxyethanol) [X] NOAEL [] LOAEL
Critical Effect:	Testicular effects (2-methoxyethanol)
Species:	Rabbit and rat
Route of Exposure:	Inhalation
Duration:	13 weeks
Uncertainty Factor:	1,000: 10 for protection of sensitive humans 10 for use of a subchronic study 10 to account for extrapolation from animals to humans and for database deficiencies
Modifying Factor:	1

Summary of Study:

Groups of New Zealand white rabbits (5/sex/dose) and Sprague-Dawley rats (10/sex/dose) were exposed to 0, 30, 100, or 300 ppm 2-methoxyethanol (0, 93, 311, or 934 mg/m³) 6 h/d, 5 d/wk for 13 weeks (Miller et al., 1983). The duration-adjusted exposure concentrations were 0, 17, 56, and 167 mg/m³, respectively. Toxicity was assessed by clinical observations, body and organ weights, hematology, clinical chemistry, urinalysis (rats only), and gross and histopathological examination of major organs including respiratory tract, heart, liver, kidney, bone marrow, testes, uterus, and ovaries.

Effects reported in both sexes of rabbits exposed to 300 ppm included reduced body weight, hematological changes (pancytopenia), lymphoid tissue atrophy (thymus), and a significant decrease in testicular weight with small flaccid testes in the males. A slight to moderate decrease in testes size was also reported in 2/5 and 4/5 male rabbits exposed to 30 and 100 ppm, respectively. Microscopic lesions included degenerative changes in the germinal epithelium of the testes in 3/3, 3/5, and 1/5 male rabbits exposed to 300, 100, and 30 ppm, respectively. The decrease in testes weight was considered to be concentration-dependent in the male rabbits. No effects on the reproductive organs of the female rabbits were found. Thymus weights were significantly decreased in both sexes exposed to 300 ppm. Based upon the testicular effects in

rabbits, a NOAEL of 30 ppm ($\text{NOAEL}_{\text{ADJ}} = 17 \text{ mg/m}^3$) and a LOAEL of 100 ppm ($\text{LOAEL}_{\text{ADJ}} = 56 \text{ mg/m}^3$) were identified.

The authors reported a significant decrease in body weight in the male rats exposed to 300 ppm and in the females exposed to 100 ppm or more. Effects reported in both sexes of rats exposed to 300 ppm included hematological changes (pancytopenia), lymphoid tissue atrophy, a decrease in liver weight, and changes in clinical chemistry parameters. In the 300 ppm group (male and female rats), the mean values for total serum protein, albumin, and globulins were lower than the control values. A significant decrease in testicular weight and small flaccid testes were also reported in the male rats exposed to 300 ppm. Microscopic examination showed moderate to severe degeneration of the germinal epithelium in the seminiferous tubules at the highest exposure. There were no microscopic changes in the testes in the animals exposed to 100 or 30 ppm. The authors found no effects in the reproductive organs of the female rats.

Based on the observed testicular effects at 100 ppm (Miller et al., 1983), a NOAEL of 30 ppm for 2-methoxyethanol in the rabbit was identified ($\text{NOAEL}_{\text{HEC}} = 17 \text{ mg/m}^3$). The NOAEL was adjusted for intermittent exposure. A $\text{NOAEL}_{\text{HEC}}$ was calculated as per EPA's inhalation dosimetry methodology (1994), using equation 4-48a (category 3 - extrarrespiratory effects). An uncertainty factor of 1000 was applied (10 for protection of sensitive humans, 10 for use of a subchronic study, and 10 to account for extrapolation from animals to humans and for database deficiencies). An RfC of 0.02 mg/m^3 was calculated for 2-methoxyethanol (U.S. EPA, 1998).

Calculations and Conversion Factors:

Dose levels are:

30 ppm 2-methoxyethanol $\times (76.09/24.45) = 93 \text{ mg/m}^3$ 2-methoxyethanol; 100 ppm = 311 mg/m^3 ; 300 ppm = 934 mg/m^3 .

$$\text{NOAEL}_{\text{ADJ}} = 93 \text{ mg/m}^3 \times (6/24 \text{ hours}) \times (5/7 \text{ days}) = 17 \text{ mg/m}^3$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times \text{RGDR}$$

$$\text{NOAEL}_{\text{HEC}} = \text{NOAEL}_{\text{ADJ}} \times (\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$$

$$\text{NOAEL}_{\text{HEC}} = 17 \text{ mg/m}^3 \times 1 = 17 \text{ mg/m}^3$$

where

$\text{NOAEL}_{\text{ADJ}}$ is the adjusted NOAEL, RGDR is the regional gas dose ratio (animal:human), and $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ is the ratio of blood:gas partition coefficients; $(\text{H}_{\text{b/g}})_{\text{A}} / (\text{H}_{\text{b/g}})_{\text{H}}$ defaults to 1 where $\text{H}_{\text{b/g}}$ values are not known.

$$\text{RfC for 2-methoxyethanol} = \text{NOAEL}_{\text{HEC}} \div \text{UF} = 17 \text{ mg/m}^3 \div 1,000 = 0.02 \text{ mg/m}^3$$

$$\text{RfC (2-methoxyethanol)} \times [\text{MW (2-methoxyethanol acetate)} \div \text{MW (2-methoxyethanol)}] = \text{RfC (2-methoxyethanol acetate)}$$

$$0.02 \text{ mg/m}^3 \times (118.13/76.09) = 0.03 \text{ mg/m}^3 = \text{RfC (2-methoxyethanol acetate)}$$

Additional Information:

The RfC of 0.02 mg/m^3 for 2-methoxyethanol (U.S. EPA, 1998) is based on male reproductive effects in New Zealand rabbits. It is assumed that 2-methoxyethanol acetate would be equitoxic to 2-methoxyethanol on a molar basis, since both compounds are metabolized to methoxyacetic

acid by the alcohol dehydrogenase pathway (DOL-OSHA, 1993). The RfC for 2-methoxyethanol acetate was calculated from the RfC for 2-methoxyethanol of 0.02 mg/m³ (U.S. EPA, 1998), resulting in an RfC of 0.03 mg/m³ for 2-methoxyethanol acetate.

Strengths and Uncertainties:

The strength of the RfC is that it is based on an RfC in IRIS that has undergone Agency review. The major uncertainty of the RfC for 2-methoxyethanol acetate is that it is based on an RfC for 2-methoxyethanol and converted using the molecular weight ratio. However, it is assumed that 2-methoxyethanol acetate would be equitoxic to 2-methoxyethanol on a molar basis.

References:

- Department of Labor, Occupational Safety and Health Administration (DOL-OSHA). 1993. Federal Register: Occupational exposure to 2-methoxyethanol, 2-ethoxyethanol and their acetates (glycol ethers); Proposed rule. 58FR15526. March 23, 1993.
- Miller, R.R., J.A. Ayres, J.T. Young, and M.J. McKenna. 1983. Ethylene glycol monomethyl ether. I. Subchronic vapor inhalation study with rats and rabbits. *Fund Appl Toxicol* 3(1):49-54.
- Research Triangle Institute (RTI). 1996. Assessment of Risks from the Management of Used Solvents. U.S. Environmental Protection Agency, Office of Solid Waste, Washington, DC. July 1996. [In support of 61FR42317]
- U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.
- U.S. Environmental Protection Agency. 1996. Federal Register: Hazardous Waste Management System; Identification and Listing of Hazardous Waste; Solvents; CERCLA Hazardous Substance Designation and Reportable Quantities; Proposed Rule. 61FR42317-354. August 14, 1996.
- U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS) file for 2-methoxyethanol.

Vanadium
CAS # 7440-62-2

RfC:	0.00007 mg/m ³
Critical Study:	Zenz, C., and B.A. Berg. 1967. Human responses to controlled vanadium pentoxide exposure. <i>Arch Environ Health</i> 14:709-712.
Critical Dose:	0.06 mg vanadium/m ³ [] NOAEL [X] LOAEL
Critical Effect:	Upper respiratory symptoms (coughing)
Species:	Human
Route of Exposure:	Inhalation
Duration:	8 hours
Uncertainty Factor:	300: 10 for use of a LOAEL in a human study 10 for use of an acute study 3 for protection of sensitive human subpopulations
Modifying Factor:	1

Calculations:

$$\text{RfC} = \text{LOAEL}_{\text{ADJ}} \div \text{UF} = 0.02 \text{ mg/m}^3 \div 300 = 0.00007 \text{ mg/m}^3$$

Summary of Study:

Nine human volunteers were exposed to vanadium pentoxide at levels of 0.1, 0.25, and 1.0 mg/m³ (0.06, 0.1, and 0.6 mg vanadium/m³) for 8 hours (Zenz and Berg, 1967). Subjects exposed to 0.06 mg vanadium/m³ experienced no symptoms during or immediately after exposure. Within 24 hours, considerable mucus formed, which resulted in slight coughing that increased after 48 hours, subsided within 72 hours, and disappeared completely after 4 days. All individuals exposed to 0.1 mg vanadium/m³ developed a loose cough the following morning, while those individuals exposed to 0.6 mg vanadium/m³ developed frequent coughing that persisted for 8 days. There were no other signs of irritation, fever, or increased pulse rate.

A LOAEL of 0.06 mg/m³ was selected, since the subjects experienced slight coughing at this dose. This LOAEL was adjusted for exposure duration (0.02 mg/m³). An uncertainty factor of 300 was applied: 10 for use of a LOAEL in a human study, 10 for use of an acute study (8 hours' duration), and 3 for protection of sensitive human subpopulations, since this study involved only nine volunteers who cannot be assumed to be the most sensitive individuals to this type of effect.

Conversion Factors:

Dose levels are:

% vanadium in vanadium pentoxide = 56.02% = 60% = 0.6. $0.1 \text{ mg V}_2\text{O}_5/\text{m}^3 \times 0.6 = 0.06 \text{ mg vanadium}/\text{m}^3$; $0.25 \text{ mg V}_2\text{O}_5/\text{m}^3 = 0.15 \text{ mg vanadium}/\text{m}^3$; $1 \text{ mg V}_2\text{O}_5/\text{m}^3 = 0.6 \text{ mg vanadium}/\text{m}^3$.

$\text{LOAEL}_{\text{ADJ}} = 0.06 \text{ mg}/\text{m}^3 \times 8/24 \text{ h} = 0.02 \text{ mg}/\text{m}^3$

Additional Information:

Other human studies support the critical effect (upper respiratory symptoms, coughing) reported in the Zenz and Berg (1967) study. Workers exposed to vanadium pentoxide for durations ranging from 1 day to 6 or more years showed mild respiratory distress, such as cough, wheezing, chest pain, runny nose, or sore throat (Levy et al., 1984; Musk and Tees, 1982; Orris et al., 1983; Sjoeborg, 1956; Thomas and Stiebris, 1956, as cited in ATSDR, 1992). Animal studies also support the human findings; monkeys, rats, and rabbits experienced respiratory effects from inhalation exposure to vanadium pentoxide (Knecht et al., 1985; Lee and Gillies, 1986; Sjoeborg, 1950; as cited in ATSDR, 1992).

Strengths and Uncertainties:

The major strength of the RfC is that it was based on human data with a critical effect that has been reported in numerous other studies. The major uncertainty to the RfC is the short duration (8 hours) and the limited number of individuals (9) in the Zenz and Berg (1967) study. In addition, a NOAEL was not identified in the study.

References:

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological profile for vanadium. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.

Knecht, E.A., W.J. Moorman, J.C. Clark, et al. 1985. Pulmonary effects of acute vanadium pentoxide inhalation in monkeys. *Am Rev Respir Dis* 132:1181-1185 (as cited in ATSDR, 1992).

Lee, K.P., and P.J. Gillies. 1986. Pulmonary response and intrapulmonary lipids in rats exposed to bismuth orthovanadate dust by inhalation. *Environ Res* 40:115-135 (as cited in ATSDR, 1992).

Levy, B.S., L. Hoffman, and S. Gottsegen. 1984. Boilmakers' bronchitis. *J Occup Med* 26: 567-570 (as cited in ATSDR, 1992).

Musk, A.W., and J.G. Tees. 1982. Asthma caused by occupational exposure to vanadium compounds. *Med J Aust* 1:183-184 (as cited in ATSDR, 1992).

Orris, P., J. Cone, and S. McQuilkin. 1983. Health hazard evaluation report HETA 80-096-1359, Bloomington, IL. Washington, DC: U.S. Department of Health and Human Services, National Institute of Occupational Safety and Health. NTIS-PB85-163574 (as cited in ATSDR, 1992).

Sjoeberg, S.G. 1950. Vanadium pentoxide dust - a clinical and experimental investigation on its effects after inhalation. *Acta Med Scand Supp.* 238:1-188 (as cited in ATSDR, 1992).

Sjoeberg, S.G. 1956. Vanadium dust, chronic bronchitis, and possible risk of emphysema. *Acta Med Scand* 154:381-386 (as cited in ATSDR, 1992).

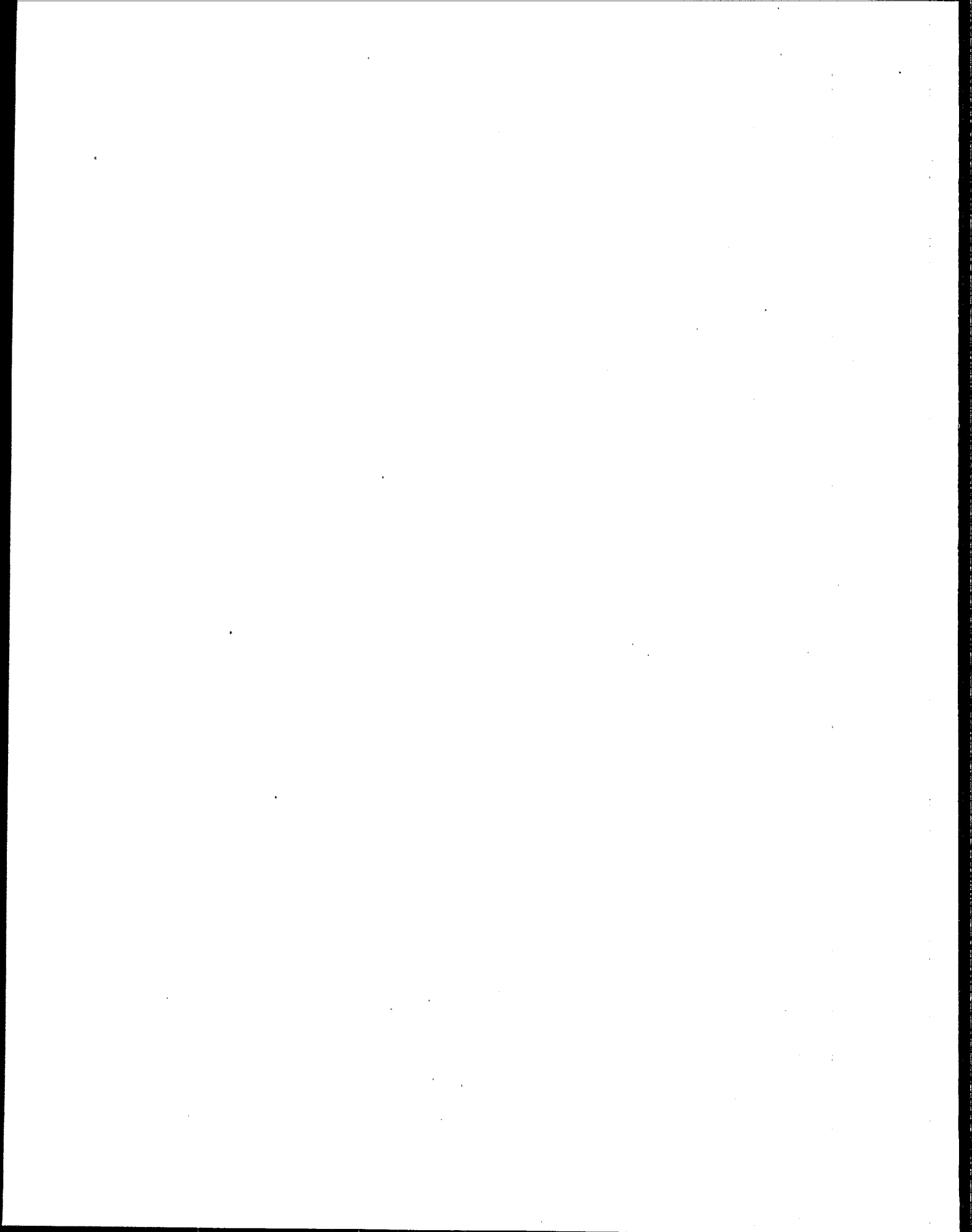
Thomas, D.L., and K. Stiebris. 1956. Vanadium poisoning in industry. *The Medical Journal of Australia* 1:607-609 (as cited in ATSDR, 1992).

U.S. Environmental Protection Agency. 1994. Methods for derivation of inhalation reference concentrations and application of inhalation dosimetry. Research Triangle Park, NC: Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Office of Research and Development, U.S. EPA. EPA/600/8-90-066F.

Zenz, C., and B.A. Berg. 1967. Human responses to controlled vanadium pentoxide exposure. *Arch Environ Health* 14:709-712.

CARCINOGENS

**DERIVATION OF INHALATION UNIT RISK FACTORS
AND CANCER SLOPE FACTORS**



Bromodichloromethane
CAS #75-27-4

Inhalation Unit Risk Factor:	1.8E-05 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	6.2E-02 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Tubular cell adenoma and tubular cell adenocarcinoma
Species:	Mice
Route of Exposure:	Gavage, corn oil
Duration:	2 years

Basis for Toxicity Values:

EPA has not developed an inhalation reference concentration (RfC) for bromodichloromethane. An oral reference dose (RfD) value of 0.02 mg/kg/d, based on a chronic gavage study in mice for renal cytomegaly is available on IRIS for bromodichloromethane (U.S. EPA, 1998).

Based on inadequate human data and sufficient evidence of carcinogenicity in animals, EPA considers bromodichloromethane a probable human carcinogen (Class B2) by the oral route and has calculated an oral cancer slope factor (CSF) of 0.062 ($\text{mg}/\text{kg}/\text{d}$)⁻¹ for the substance. In a National Toxicology Program (NTP) study, 2-year gavage administration of bromodichloromethane to both sexes of F344/N rats and B6C3F1 resulted in compound-related statistically significant increases in tumors of the kidney in male mice, the liver in female mice, and the kidney and large intestine in male and female rats (NTP, 1987, as cited in U.S. EPA, 1998).

In male mice, the incidences of tubular cell adenomas and the combined incidence of tubular cell adenomas and adenocarcinomas of the kidneys were significantly increased in the high-dose animals. In female mice, there were significant increases of hepatocellular adenomas and hepatocellular carcinomas. The combined incidence of hepatocellular adenomas or carcinomas in vehicle control, low-dose, and high-dose groups were 3/50, 18/48, and 29/50, respectively.

In male and female rats, the incidences of tubular cell adenomas, adenocarcinomas, and the combined incidence of adenomas and adenocarcinomas of the kidneys were statistically significantly increased only in the high-dose groups. The combined incidence of tubular cell adenomas or adenocarcinomas in vehicle control, low-dose, and high-dose groups were 0/50, 1/49, and 13/50 for males and 0/50, 1/50, and 15/50 for females, respectively.

Tumors of the large intestines, namely adenocarcinomas and adenomatous polyps, were significantly increased in male rats in a dose-dependent manner. These large intestinal tumors, however, were observed only in high-dose female rats (adenocarcinomas 0/46, 0/50, 6/47; adenomatous polyps 0/46, 0/50, 7/47 in the vehicle control, low-dose and high-dose groups, respectively). The combined incidence of large intestine adenocarcinomas and/or adenomatous polyps in vehicle control, low-dose, and high-dose groups were 0/50, 13/49, and 45/50 for males and 0/46, 0/50, and 12/47 for females. The combined tumor incidences in the large intestine and

kidney in male and female rats at control, low dose, and high dose were 0/50, 13/49, 46/50 and 0/46, 1/50, 24/48, respectively. Under the conditions of this bioassay, the NTP concluded there was clear evidence of carcinogenicity of bromodichloromethane in male and female F344/N rats and B6C3F1 mice (U.S. EPA, 1998).

The mechanism for the carcinogenicity of bromodichloromethane appears to be genotoxic carcinogenesis, independent of liver activation and, hence, route-independent. In one genotoxicity assay, bromodichloromethane was mutagenic in *Salmonella typhimurium* strain TA100 in the absence of liver homogenate in a vapor phase test performed in a desiccator. Positive results for mutagenicity were reported for bromodichloromethane in other *S. typhimurium* assays in which the TA100 and TA1537 strains were used without rat liver homogenate activation. Bromodichloromethane also induced weak mutagenic effects in *Saccharomyces cerevisiae* strains D7 and XV185-14C in the absence of liver homogenate (U.S. EPA, 1998; HSDB, 1998).

Thus, inhalation exposure to bromodichloromethane is likely to lead to carcinogenic consequences not dissimilar from that from oral exposure. Therefore, in accordance with current EPA guidelines, it is considered appropriate to calculate an inhalation unit risk factor for bromodichloromethane from the oral CSF listed for that substance in IRIS (U.S. EPA, 1994, 1996).

Calculations:

$$\text{URF} = \text{CSF} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{day} = \\ 0.062 (\text{mg}/\text{kg}/\text{d})^{-1} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 1.8\text{E-}05(\mu\text{g}/\text{m}^3)^{-1}$$

where

70 kg = default adult human body weight

20 m³ = default adult human daily rate of inhalation

Calculations assume 100% absorption.

Additional Information:

Inhalation CSFs are often derived from oral data. Of the 51 chemicals currently listed in IRIS (U.S. EPA, 1998) and HEAST (U.S. EPA, 1997) that have both an oral and inhalation CSF, about 60% of the inhalation CSFs were derived from oral studies and are identical or essentially identical to the oral CSF (see Table E-1, Figure E-1). In at least one case (benzene), the oral CSF was based on inhalation data resulting in identical values for both routes of exposure. In most cases (>75%) where an inhalation CSF was derived from an inhalation study, the inhalation CSF was lower than the corresponding oral CSF. Therefore, use of an oral CSF as an interim inhalation CSF appears reasonable and is unlikely to result in underestimating risk.

References:

Hazardous Substances Databank (HSDB): Bromodichloromethane. 1998. Online database. National Library of Medicine, Bethesda, MD.

National Toxicology Program (NTP). 1987. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Bromodichloromethane (CAS no. 75-27-4) in F344/N Rats and B6C3F1 Mice (gavage studies). NTP Tech. Report Series No.321. U.S. Dept. Health and Human Services, Public Health Service, National Institute of Health (as cited in U.S. EPA, 1998).

U.S. Environmental Protection Agency. 1994. Provisional Guidance for the Qualitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Prepared by the Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH, for the Office of Research and Development, Cincinnati, OH. EPA/600/R-93.

U.S. Environmental Protection Agency. 1996. Proposed Guidelines for Carcinogen Risk Assessment. Office of Research and Development. Washington, DC. EPA/600/P-92/003C.

U.S. Environmental Protection Agency. 1997. Health Effects Assessment Summary Tables (HEAST), FY 1997 Update. Office of Emergency and Remedial Response, Washington, DC. EPA-540-R-97-036.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). Bromodichloromethane. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

Chlorodibromomethane
CAS #124-48-1

Inhalation Unit Risk Factor:	2.4E-05 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	8.4E-02 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Hepatocellular adenoma or carcinoma
Species:	Mice
Route of Exposure:	Gavage
Duration:	2 years

Basis for Toxicity Values:

EPA has not developed an inhalation reference concentration (RfC) for chlorodibromomethane. An oral reference dose (RfD) value of 0.02 mg/kg/d, based on a subchronic gavage study in rats for hepatic lesions is available on IRIS for chlorodibromomethane (U.S. EPA, 1998)

Based on inadequate human data and limited evidence of carcinogenicity in animals, EPA considers chlorodibromomethane a possible human carcinogen (Class C) by the oral route and has calculated an oral cancer slope factor (CSF) of 0.084 ($\text{mg}/\text{kg}/\text{d}$)⁻¹ for the substance. In the study, 2-year gavage administration of chlorodibromomethane to both sexes of B6C3F1 mice caused increased incidence of adenomas and carcinomas in female mice and a significantly increased incidence of hepatocellular carcinomas in high-dose male mice (NTP, 1985, as cited in U.S. EPA, 1998). Drinking water administration of chlorodibromomethane to both sexes of CBAxCS7B1/6 mice also resulted in significantly increased incidence of tumors (U.S. EPA, 1998).

The mechanism for the carcinogenicity of chlorodibromomethane appears to be genotoxic carcinogenesis, independent of liver activation and, hence, route-independent. In one genotoxicity assay, chlorodibromomethane produced reverse mutations in *Salmonella typhimurium* strain TA100 in a vapor-phase test performed in a desiccator. Positive results for gene conversion in *Saccharomyces cerevisiae* strain D4 without, but not with, hepatic homogenates, and negative results for mutation in strain XV185-14C both with and without hepatic homogenates have been reported for chlorodibromomethane. In others tests, chlorodibromomethane produced sister chromatid exchange in cultured human lymphocytes and in bone marrow cells of mice treated orally (U.S. EPA, 1998; HSDB, 1998).

Thus, inhalation exposure to chlorodibromomethane is likely to lead to carcinogenic consequences not dissimilar from that from oral exposure. Therefore, in accordance with current EPA guidelines, it is considered appropriate to calculate an inhalation unit risk factor for chlorodibromomethane from the oral CSF listed for that substance in IRIS (U.S. EPA, 1994, 1996).

Calculations:

$$\text{URF} = \text{CSF} \times 1 \text{ mg}/1,000 \text{ } \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = \\ 0.084 \text{ (mg/kg/d)}^{-1} \times 1 \text{ mg}/1,000 \text{ } \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 2.4\text{E-}05(\mu\text{g}/\text{m}^3)^{-1}$$

where

70 kg = default adult human body weight

20 m³ = default adult human daily rate of inhalation

Calculations assume 100% absorption.

Additional Information:

Inhalation CSFs are often derived from oral data. Of the 51 chemicals currently listed in IRIS (EPA, 1998) and HEAST (U.S. EPA, 1997) that have both an oral and inhalation CSF, about 60% of the inhalation CSFs were derived from oral studies and are identical or essentially identical to the oral CSF (see Table E-1, Figure E-1). In at least one case (benzene), the oral CSF was based on inhalation data resulting in identical values for both routes of exposure. In most cases (>75%) where an inhalation CSF was derived from an inhalation study, the inhalation CSF was lower than the corresponding oral CSF. Therefore, use of an oral CSF as an interim inhalation CSF appears reasonable and is unlikely to result in underestimating risk.

References:

Hazardous Substances Databank (HSDB): Chlorodibromomethane. 1998. Online database. National Library of Medicine, Bethesda, MD.

National Toxicology Program (NTP). 1985. Toxicology and Carcinogenesis Studies of Chlorodibromomethane in F344/N Rats and B6C3F1 Mice (gavage studies). NTP TR282 (as cited in U.S. EPA, 1998).

U.S. Environmental Protection Agency. 1994. Provisional Guidance for the Qualitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Prepared by the Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH, for the Office of Research and Development, Cincinnati, OH.

U.S. Environmental Protection Agency. 1996. Proposed Guidelines for Carcinogen Risk Assessment. Office of Research and Development. Washington, DC. EPA/600/P-92/003C.

U.S. Environmental Protection Agency. 1997. Health Effects Assessment Summary Tables (HEAST), FY 1997 Update. Office of Emergency and Remedial Response, Washington, DC. EPA-540-R-97-036.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). Chlorodibromomethane. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

7,12-Dimethylbenz[a]anthracene
CAS # 57-97-6

Unit Risk Factor:	2.4E-02 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	8.4E+01 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Malignant angioendothelioma of the mesenteric intestine
Species:	Mouse (albino)
Route of Exposure:	Diet
Duration:	60 weeks

Basis for Toxicity Values:

There are no human data available that may be used to address the carcinogenicity of 7,12-dimethylbenz[a]anthracene (DMBA). However, DMBA belongs to a class of chemicals known as polycyclic aromatic hydrocarbons (PAHs), which are components of coal tar and incomplete combustion. Many of the PAHs have been demonstrated to be carcinogenic to rats and mice following oral exposure, skin painting, intrapulmonary injection, inhalation, subcutaneous injection, and intraperitoneal injection; however, most of these studies are not considered suitable for quantitative risk assessment. Nevertheless, the data do indicate that the carcinogenic potencies vary and that DMBA is considered one of the most potent PAHs (Pitot and Dragan, 1996).

DMBA is not listed in EPA's IRIS (U.S. EPA, 1998) or HEAST (U.S. EPA, 1997) databases and was not included in EPA's (1993) *Provisional Guidance for Quantitative Risk Assessment of PAHs*. However, the California Environmental Protection Agency (CalEPA) has developed a unit risk factor (URF) and cancer slope factor (CSF) for DMBA in support of the Air Toxics Hot Spots Program (CalEPA, 1994a, 1994b, 1997). The CalEPA URF and inhalation CSF are listed above and are recommended as interim values.

The CalEPA developed an "expedited" approach for deriving cancer potency values in order to implement Proposition 65 (Hoover et al., 1995). The expedited approach was used for DMBA. Under the expedited approach, instead of conducting a comprehensive literature review, cancer dose response data are taken from the Carcinogenic Potency Database (CPDB) (Gold and Zeiger, 1997). The linearized multistage model is automatically used to derive cancer potency estimates for low-dose exposures, and pharmacokinetic adjustments are not made.

Only one study was listed in the CPDB (Chouroulinkov et al., 1967). Female albino mice were fed DMBA for 60 weeks at a dose rate of 0.39 mg/kg/d. No tumors were reported in 40 control mice. Malignant angioendotheliomas of the intestine were reported in 49 of 75 test animals. Twenty test animals also had nonmalignant forestomach papillomas.

Additional Information:

The CPDB summarizes the results of 5,152 cancer tests on 1,298 chemicals. Carcinogenic potency estimates are presented as TD_{50} values. TD_{50} is defined as *that dose-rate in mg/kg body wt/d which, if administered chronically for the standard lifespan of the species, will halve the probability of remaining tumorless throughout that period* (Gold and Zeiger, 1997). The TD_{50} is analogous to the dose that is lethal to 50 percent of test animals (LD_{50}). A low TD_{50} indicates high potency, just as a low LD_{50} indicates high acute toxicity.

Some studies have reported high correlations between various measures of cancer potency and the maximum tolerated dose or maximum dose tested in the carcinogenicity studies (Gaylor, 1989; Krewski et al., 1993). The correlation of TD_{50} values as reported in the CPDB and inhalation CSFs derived from IRIS or HEAST was evaluated as a possible means to estimate the CSF from the TD_{50} . Forty-five chemicals were identified that had both a TD_{50} and an inhalation CSF (see Table E-2, Figure E-2). The correlation coefficient for the regression is 0.95. The TD_{50} reported for DMBA is 0.084 mg/kg/d (Gold and Zeiger, 1997). Based on a linear regression of $\log TD_{50}$ as the independent variable and $\log (1/CSF)$ as the dependent variable, an inhalation CSF of 55 (mg/kg/d)⁻¹ and a URF of 1.6E-02 ($\mu\text{g}/\text{m}^3$)⁻¹ are predicted. These values are in close agreement with the CalEPA values of 84 (mg/kg/d)⁻¹ and 2.4E-02($\mu\text{g}/\text{m}^3$)⁻¹, respectively.

References:

- California Environmental Protection Agency (CalEPA). 1994a. Benzo[a]pyrene as a Toxic Air Contaminant. Executive Summary. California Air Resources Board, Office of Environmental Health Hazard Assessment, Berkeley, CA.
- California Environmental Protection Agency (CalEPA). 1994b. Benzo[a]pyrene as a Toxic Air Contaminant. Part B Health Effects of Benzo(a)pyrene. California Air Resources Board, Office of Environmental Health Hazard Assessment, Berkeley, CA.
- California Environmental Protection Agency (CalEPA). 1997. Air Toxics Hot Spots Program Risk Assessment Guidelines: Technical Support Document for Determining Cancer Potency Factors. Draft for Public Comment. Office of Environmental Health Hazard Assessment.
- Chouroulinkov, I., A. Gentil, and M. Guerin. 1967. Étude de l'activité carcinogène du 9,10-diméthyl-benzanthracène et du 3,4-benzopyrène administrés par voie digestive. *Bull Cancer* 54:67-78 (as cited in Gold and Zeiger, 1997).
- Gaylor, D.W. 1989. Preliminary estimates of the virtually safe dose for tumors obtained from the maximum tolerated dose. *Regulatory Toxicology and Pharmacology* 9:1-18.
- Gold, L.S., and E. Zeiger (eds). 1997. *Handbook of Carcinogenic Potency and Genotoxicity Databases*. Boca Raton, FL: CRC Press, 754 pp.
- Hoover, S.M., L. Zeise, W.S. Pease, et al. 1995. Improving the regulation of carcinogens by expediting cancer potency estimation. *Risk Analysis* 15(2):267-280.

Krewski, D., D.W. Gaylor, A.P. Soms, and M. Szyszkowicz. 1993. An overview of the report: Correlation between carcinogenic potency and the maximum tolerated dose: Implications for risk assessment. *Risk Analysis* 13(4):383-398.

Pitot, H.C., III, and Y.P. Dragan. 1996. Chemical Carcinogenesis. In: *Casarett & Doull's Toxicology the Basic Science of Poisons*. 5th edition. C.D. Klaassen (ed). New York: McGraw-Hill, pp. 202-203.

U.S. Environmental Protection Agency. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. EPA/600/R-93/089.

U.S. Environmental Protection Agency. 1997. Health Effects Assessment Summary Tables (HEAST), FY 1997 Update. Office of Emergency and Remedial Response, Washington, DC. EPA-540-R-97-036.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

2,4-Dinitrotoluene

CAS #121-14-2

Unit Risk Factor:	1.9E-04 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	6.8E-01 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Hepatocellular carcinoma, liver neoplastic nodules, benign and malignant mammary gland tumors.
Species:	Female Sprague-Dawley rats
Route of exposure:	Diet
Duration:	2 years

Basis for Toxicity Values:

There are no human data available that may be used to address the carcinogenicity of 2,4-dinitrotoluene. 2,4-Dinitrotoluene is not listed in EPA's IRIS (U.S. EPA, 1998) or HEAST (U.S. EPA, 1997) databases. However, an oral CSF of 0.68 ($\text{mg}/\text{kg}/\text{d}$)⁻¹ is available in IRIS for a mixture of 2,4- and 2,6-dinitrotoluene. The mixture was 98% 2,4-dinitrotoluene and 2% 2,6-dinitrotoluene. The oral CSF for the mixture is proposed as an interim value for the inhalation CSF for 2,4-dinitrotoluene.

Inhalation CSFs are often derived from oral data. Of the 51 chemicals currently listed in IRIS and HEAST that have both an oral and inhalation CSF, about 60% of the inhalation CSFs were derived from oral studies and are identical or essentially identical to the oral CSF (see Table E-1, Figure E-1). In at least one case (benzene), the oral CSF was based on inhalation data resulting in identical values for both routes of exposure. In most cases (>75%) where an inhalation CSF was derived from an inhalation study, the inhalation CSF was lower than the corresponding oral CSF. Therefore, use of an oral CSF as an interim inhalation CSF appears reasonable and is unlikely to result in underestimating risk.

Dose-Response Data:

The oral CSF listed in HEAST was based on a study by Ellis et al. (1979). Sprague-Dawley rats were fed dietary concentrations of 0, 15, 100, and 700 ppm and Swiss mice were fed 0, 100, 700, and 5,000 ppm for 2 years. Mortality was high in all treatment groups. A statistically significant increase in liver tumors was observed in both male and female rats and a statistically significant increase in benign mammary gland tumors was observed in female rats. In addition, an increased incidence of kidney tumors was observed in the mid-dose male mice. Data used to derive the CSF were based on liver and mammary tumors in female rats and are presented below as reported in IRIS.

Administered Dose (ppm)	Human Equivalent Dose (mg/kg/d)	Tumor Incidence
0	0	11/23
15	0.129	12/35
100	0.927	17/27
700	7.557	34/35

Calculations:

$$\text{URF} = \text{CSF} \times 1 \text{ mg}/1,000 \text{ } \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} =$$

$$0.68 \text{ (mg/kg/d)}^{-1} \times 1 \text{ mg}/1,000 \text{ } \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 1.9\text{E-}04(\mu\text{g}/\text{m}^3)^{-1}$$

where

70 kg = default adult human body weight

20 m³ = default adult human daily rate of inhalation

Calculations assume 100% absorption.

Additional Information:

The California Environmental Protection Agency (CalEPA) adopted a URF of 8.9E-05 (μg/m³)⁻¹ and an inhalation CSF of 3.1E-01 (mg/kg/d)⁻¹ for practical grade 2,4-dinitrotoluene based on a potency factor derived by EPA (U.S. EPA, 1987) (CalEPA, 1997). These values were based on a feeding study using Sprague-Dawley rats (Lee et al., 1978). Liver and mammary tumors in female rats were used to develop the CSF and results were very similar to the Ellis et al. (1979) study discussed above.

The Carcinogenic Potency Database (CPDB) summarizes the results of 5,152 cancer tests on 1,298 chemicals (Gold and Zeiger, 1997). Carcinogenic potency estimates are presented as TD₅₀ values. TD₅₀ values are defined as *that dose-rate in mg/kg body wt/day which, if administered chronically for the standard lifespan of the species, will halve the probability of remaining tumorless throughout that period* (Gold and Zeiger, 1997). The TD₅₀ is analogous to the dose that is lethal to 50% of test animals (LD₅₀). A low TD₅₀ indicates high potency, just as a low LD₅₀ indicates high acute toxicity.

Some studies have reported high correlations between various measures of cancer potency and the maximum tolerated dose or maximum dose tested in the carcinogenicity studies (Gaylor, 1989; Krewski et al., 1993). The correlation of TD₅₀s as reported in the CPDB and inhalation CSFs derived from IRIS or HEAST was evaluated as a possible means to estimate the CSF from the TD₅₀. Forty-five chemicals were identified that had both a TD₅₀ and an inhalation CSF (see Table E-2, Figure E-2). The correlation coefficient for the regression is 0.95. The reported TD₅₀ is 9.35 mg/kg/d (Gold and Zeiger, 1997). Based on a linear regression of log TD₅₀ as the independent variable and log (1/CSF) as the dependent variable, an inhalation CSF of 0.53 (mg/kg/d)⁻¹ and a URF of 1.5E-04 (μg/m³)⁻¹ are predicted. These values are in close agreement

with the oral CSF listed in IRIS for a mixture of 2,4- and 2,6-dinitrotoluene and the CalEPA values.

References:

California Environmental Protection Agency (CalEPA). 1997. Air Toxics Hot Spots Program Risk Assessment Guidelines: Technical Support Document for Determining Cancer Potency Factors. Draft for Public Comment. Office of Environmental Health Hazard Assessment.

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Lee, C.C., H.V. Ellis, J.J. Kowalski, et al. 1978. Mammalian toxicity of munition compounds. Phase II. Effects of multiple doses and Phase III. Effects of lifetime exposure. Part II. 2,4-Dinitrotoluene. U.S. Army Medical Bioengineering Research and Development Laboratory. Midwest Research Institute, Kansas City, MO. NTIS ADA 061715.

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U.S. Environmental Protection Agency. 1997. Health Effects Assessment Summary Tables (HEAST), FY 1997 Update. Office of Emergency and Remedial Response, Washington, DC. EPA-540-R-97-036.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

3-Methylcholanthrene CAS # 56-49-5

Unit Risk Factor:	2.1E-03 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	7.4E+00 ($\text{mg}/\text{kg}/\text{day}$) ⁻¹
Critical Effects:	Mammary gland adenocarcinomas
Species:	Wistar rats
Route of Exposure:	Gavage
Duration:	26 to 52 weeks

Basis for Toxicity Values:

There are no human data available that may be used to address the carcinogenicity of 3-methylcholanthrene (3-MC). However, 3-MC belongs to a class of chemicals known as polycyclic aromatic hydrocarbons (PAHs), which are components of coal tar and incomplete combustion. Many of the PAHs have been demonstrated to be carcinogenic to rats and mice following oral exposure, skin painting, intrapulmonary injection, inhalation, subcutaneous injection, and intraperitoneal injection; however, most of these studies are not considered suitable for quantitative risk assessment. Nevertheless, the data do indicate that the carcinogenic potencies vary and that 3-MC is considered one of the most potent PAHs (Pitot and Dragan, 1996).

3-MC is not listed in EPA's IRIS (U.S. EPA, 1998) or HEAST (U.S. EPA, 1997) databases and was not included in EPA's (1993) *Provisional Guidance for Quantitative Risk Assessment of PAHs*. However, the California Environmental Protection Agency (CalEPA) has developed a unit risk factor (URF) and cancer slope factor (CSF) for 3-MC in support of the Air Toxics Hot Spots Program (CalEPA, 1994a, 1994b, 1997). The CalEPA URF and inhalation CSF are listed above and recommended as interim values.

The CalEPA developed an "expedited" approach for deriving cancer potency values in order to implement Proposition 65 (Hoover et al., 1995). The expedited approach was used for 3-MC. Under the expedited approach, instead of conducting a comprehensive literature review, cancer dose response data are taken from the Carcinogenic Potency Database (CPDB) (Gold and Zeiger, 1997). The linearized multistage model is automatically used to derive cancer potency estimates for low-dose exposures, and pharmacokinetic adjustments are not made.

Fifteen studies (4 diet and 11 gavage) were listed in the CPDB (Gold and Zeiger, 1997). All of the studies included a control group and one treatment group. No tumors were reported in any of the dietary studies; however, a significant increase in tumors was reported in all of the gavage studies. Doses for the gavage studies ranged from 2.46 mg/kg/d to 12.2 mg/kg/d. Adenocarcinomas of the mammary gland were reported in nine studies and two studies identified unspecified mammary tissue tumors. Tumor incidence ranged from 67% to 100%.

Additional Information:

The CPDB summarizes the results of 5,152 cancer tests on 1,298 chemicals. Carcinogenic potency estimates are presented as TD_{50} s. TD_{50} s are defined as *that dose-rate in mg/kg body wt/day which, if administered chronically for the standard lifespan of the species, will halve the probability of remaining tumorless throughout that period* (Gold and Zeiger, 1997). The TD_{50} is analogous to the dose that is lethal to 50% of test animals (LD_{50}). A low TD_{50} indicates high potency, just as a low LD_{50} indicates high acute toxicity.

Some studies have reported high correlations between various measures of cancer potency and the maximum tolerated dose or maximum dose tested in the carcinogenicity studies (Gaylor, 1989; Krewski et al., 1993). The correlation of TD_{50} s as reported in the CPDB and inhalation CSFs derived from IRIS or HEAST was evaluated as a possible means to estimate the CSF from the TD_{50} . Forty-five chemicals were identified that had both a TD_{50} and an inhalation CSF (see Table E-2, Figure E-2). The correlation coefficient for the regression is 0.95. The TD_{50} reported for 3-MC is 0.491 mg/kg/d (Gold and Zeiger, 1997). Based on a linear regression of $\log TD_{50}$ as the independent variable and $\log (1/\text{CSF})$ as the dependent variable, an inhalation CSF of $9.6 (\text{mg/kg/d})^{-1}$ and a URF of $2.7\text{E-}03 (\mu\text{g/m}^3)^{-1}$ are predicted. These values are in close agreement with the CalEPA values of $7.4 (\text{mg/kg/d})^{-1}$ and $2.1\text{E-}03 (\mu\text{g/m}^3)^{-1}$, respectively.

References:

- California Environmental Protection Agency (CalEPA). 1994a. Benzo[a]pyrene as a Toxic Air Contaminant. Executive Summary. California Air Resources Board, Office of Environmental Health Hazard Assessment, Berkeley, CA.
- California Environmental Protection Agency (CalEPA). 1994b. Benzo[a]pyrene as a Toxic Air Contaminant. Part B Health Effects of Benzo(a)pyrene. California Air Resources Board, Office of Environmental Health Hazard Assessment, Berkeley, CA.
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Pitot, H.C., III, and Y.P. Dragan. 1996. Chemical Carcinogenesis. In: *Casarett & Doull's Toxicology the Basic Science of Poisons*. 5th edition. C.D. Klaassen (ed). New York: McGraw-Hill, pp. 202-203.

U.S. Environmental Protection Agency. 1993. Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons. Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH. EPA/600/R-93/089.

U.S. Environmental Protection Agency. 1997. Health Effects Assessment Summary Tables, FY 1997 Update. Office of Emergency and Remedial Response, Washington, DC. EPA-540-R-97-036.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

o-Toluidine (2-Methylaniline)
CAS # 95-53-4

Unit Risk Factor:	6.9E-05 ($\mu\text{g}/\text{m}^3$) ⁻¹
Slope Factor:	2.4E-01 ($\text{mg}/\text{kg}/\text{d}$) ⁻¹
Critical Effects:	Skin fibromas - also increased incidence of other tumor types including sarcomas, mesotheliomas, carcinomas, hemangiosarcomas, and hepatocellular carcinomas of various tissues.
Species:	F-344 rats and B6C3F1 mice
Route of Exposure:	Diet
Duration:	2 years

Basis for Toxicity Values:

There is limited evidence that o-toluidine is carcinogenic in humans; however, data are inadequate for a quantitative risk assessment (U.S. EPA, 1987). o-Toluidine is not listed in EPA's IRIS (U.S. EPA, 1998) but an oral CSF is included in HEAST (U.S. EPA, 1997). The oral CSF of 2.4E-01 $\text{mg}/\text{kg}/\text{d}$ is proposed as an interim value for the inhalation CSF.

Inhalation CSFs are often derived from oral data. Of the 51 chemicals currently listed in IRIS and HEAST that have both an oral and inhalation CSF, about 60% of the inhalation CSFs were derived from oral studies and are identical or essentially identical to the oral CSF (see Table E-1, Figure E-1). In at least one case (benzene), the oral CSF was based on inhalation data resulting in identical values for both routes of exposure. In most cases (>75%) where an inhalation CSF was derived from an inhalation study, the inhalation CSF was lower than the corresponding oral CSF. Therefore, use of an oral CSF as an interim inhalation CSF appears reasonable and is unlikely to result in underestimating risk.

Dose-Response Data:

The oral CSF listed in HEAST was based on a study by Hecht et al. (1982). Groups of 30 male F344 rats were fed dietary concentrations of 0 or 4,000 ppm o-toluidine hydrochloride for 73 weeks followed by 20 weeks of observation. An increased incidence of skin fibromas, mammary fibroadenomas, spleen fibromas, and peritoneal sarcomas was reported. Skin fibromas gave the greatest response and were used to derive the CSF. The data are summarized below as reported in U.S. EPA (1987).

Experimental Dose o-Toluidine-HCl (mg/rat/d)	Transformed Dose o-Toluidine (mg/kg/d)	Incidence
0	0	1/27
62	80	25/30

Calculations:

$$\text{URF} = \text{CSF} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} =$$

$$0.24 (\text{mg}/\text{kg}/\text{d})^{-1} \times 1 \text{ mg}/1,000 \mu\text{g} \times 1/70 \text{ kg} \times 20 \text{ m}^3/\text{d} = 6.9\text{E-}05(\mu\text{g}/\text{m}^3)^{-1}$$

where

70 kg = default adult human body weight

20 m³ = default adult human daily rate of inhalation

Calculations assume 100% absorption.

Additional Information:

The National Cancer Institute (NCI) also has conducted a cancer bioassay of o-toluidine hydrochloride (NCI, 1979). F344 rats were fed diets containing 0, 3,000, and 6,000 ppm and B6C3F₁ mice were fed diets containing 0, 1,000, and 3,000 ppm for 2 years. Multiple site sarcomas, subcutaneous fibromas, and multiple site mesotheliomas were observed in male rats. Female rats had multiple site sarcomas, mammary fibroadenomas, splenic sarcomas, and urinary bladder carcinomas. Multiple site hemangiosarcomas were seen in male mice and hepatocellular carcinomas and adenomas were seen in female mice. U.S. EPA (1987) reported that the Hecht et al. (1982) study was selected over the NCI (1979) study because the former resulted in a higher cancer potency estimate.

References:

Hecht, S.S., K. El-Bayoumy, A. Rivenson, and E. Fiala. 1982. Comparative carcinogenicity of o-toluidine hydrochloride and o-nitrosotoluene in F-344 Rats. *Cancer Letters* 16:103-108.

National Cancer Institute (NCI). 1979. Bioassay of o-Toluidine Hydrochloride for Possible Carcinogenicity. TR-153. Bethesda, MD.

U.S. Environmental Protection Agency. 1987. Health and Environmental Effects Profile for 2-Methylaniline and 2-Methylaniline Hydrochloride. Office of Health and Environmental Assessment, Cincinnati, OH. EPA/600/X-87/092.

U.S. Environmental Protection Agency. 1997. Health Effects Assessment Summary Tables (HEAST), FY 1997 Update. Office of Emergency and Remedial Response, Washington, DC. EPA-540-R-97-036.

U.S. Environmental Protection Agency. 1998. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.

**Table E-1. Correlation of Oral and Inhalation Cancer Slope Factors
Reported in IRIS and HEAST**

CAS #	Chemical	Oral CSF	Inh CSF	log Oral CSF	log Inh. CSF
79-06-1	Acrylamide	4.5	4.5	0.6532	0.6532
107-13-1	Acrylonitrile	0.54	0.24	-0.2676	-0.6198
309-00-2	Aldrin	17	17	1.2304	1.2304
140-57-8	Aramite	0.025	0.025	-1.6021	-1.6021
7440-38-2	Arsenic	1.5	15	0.1761	1.1761
103-33-3	Azobenzene	0.11	0.11	-0.9586	-0.9586
71-43-2	Benzene	0.029	0.029	-1.5376	-1.5376
92-87-5	Benzidine	230	235	2.3617	2.3711
7440-41-7	Beryllium	4.3	8.4	0.6335	0.9243
111-44-4	Bis(2-chloroethyl)ether	1.1	1.16	0.0414	0.0645
542-88-1	Bis(chloromethyl)ether	220	217	2.3424	2.3365
108-60-1	Bis(2-chloro-1-methylethyl)ether	0.07	0.035	-1.1549	-1.4559
75-25-2	Bromoform	0.0079	0.0039	-2.1024	-2.4145
56-23-5	Carbon tetrachloride	0.13	0.053	-0.8861	-1.2757
57-74-9	Chlordane	1.3	1.3	0.1139	0.1139
510-15-6	Chlorobenzilate	0.27	0.27	-0.5686	-0.5686
67-66-3	Chloroform	0.0061	0.08	-2.2147	-1.0969
74-87-3	Chloromethane	0.013	0.0063	-1.8861	-2.2007
50-29-3	DDT	0.34	0.34	-0.4685	-0.4685
96-12-8	1,2-Dibromo-3-chloropropane	1.4	0.0024	0.1461	-2.6162
106-93-4	1,2-Dibromoethane	85	0.77	1.9294	-0.1135
107-06-2	1,2-Dichloroethane	0.091	0.091	-1.0410	-1.0410
75-35-4	1,1-Dichloroethylene	0.6	0.175	-0.2218	-0.7570
542-75-6	1,3-Dichloropropene	0.18	0.13	-0.7447	-0.8861
60-57-1	Dieldrin	16	16	1.2041	1.2041
122-66-7	1,2-Diphenylhydrazine	0.8	0.77	-0.0969	-0.1135
106-89-8	Epichlorohydrin	0.0099	0.0042	-2.0044	-2.3768
75-21-8	Ethylene oxide	1.02	0.35	0.0086	-0.4559
319-84-6	HCH alpha	6.3	6.3	0.7993	0.7993
319-85-7	HCH beta	1.8	1.8	0.2553	0.2553
608-73-1	HCH tech.	1.8	1.8	0.2553	0.2553
76-44-8	Heptachlor	4.5	4.5	0.6532	0.6532
1024-57-3	Heptachlor epoxide	9.1	9.1	0.9590	0.9590
118-74-1	Hexachlorobenzene	1.6	1.6	0.2041	0.2041
87-68-3	Hexachlorobutadiene	0.078	0.077	-1.1079	-1.1135
67-72-1	Hexachloroethane	0.014	0.014	-1.8539	-1.8539

Table E-1. (continued)

CAS #	Chemical	Oral CSF	Inh CSF	log Oral CSF	log Inh. CSF
(continued)					
302-01-2	Hydrazine	3	17.1	0.4771	1.2330
75-09-2	Methylene chloride	0.0075	0.0016	-2.1249	-2.7959
101-14-4	4,4'-Methylenebis(2-chloroaniline)	0.13	0.13	-0.8861	-0.8861
924-16-3	N-Nitrosodi-n-butylamine	5.4	5.6	0.7324	0.7482
55-18-5	N-Nitrosodiethylamine	150	151	2.1761	2.1790
62-75-9	N-Nitrosodimethylamine	51	49	1.7076	1.6902
930-55-2	N-Nitrosopyrrolidine	2.1	2.13	0.3222	0.3284
1336-36-3	PCBs	2	0.4	0.3010	-0.3979
75-56-9	Propylene oxide	0.24	0.013	-0.6198	-1.8861
630-20-6	1,1,1,2,-Tetrachloroethane	0.026	0.026	-1.5850	-1.5850
79-34-5	1,1,2,2,-Tetrachloroethane	0.2	0.2	-0.6990	-0.6990
8001-35-2	Toxaphene	1.1	1.1	0.0414	0.0414
79-00-5	1,1,2-Trichloroethane	0.057	0.056	-1.2441	-1.2518
88-06-2	2,4,6,-Trichlorophenol	0.011	0.011	-1.9586	-1.9586
75-01-4	Vinyl chloride	1.9	0.3	0.2788	-0.5229

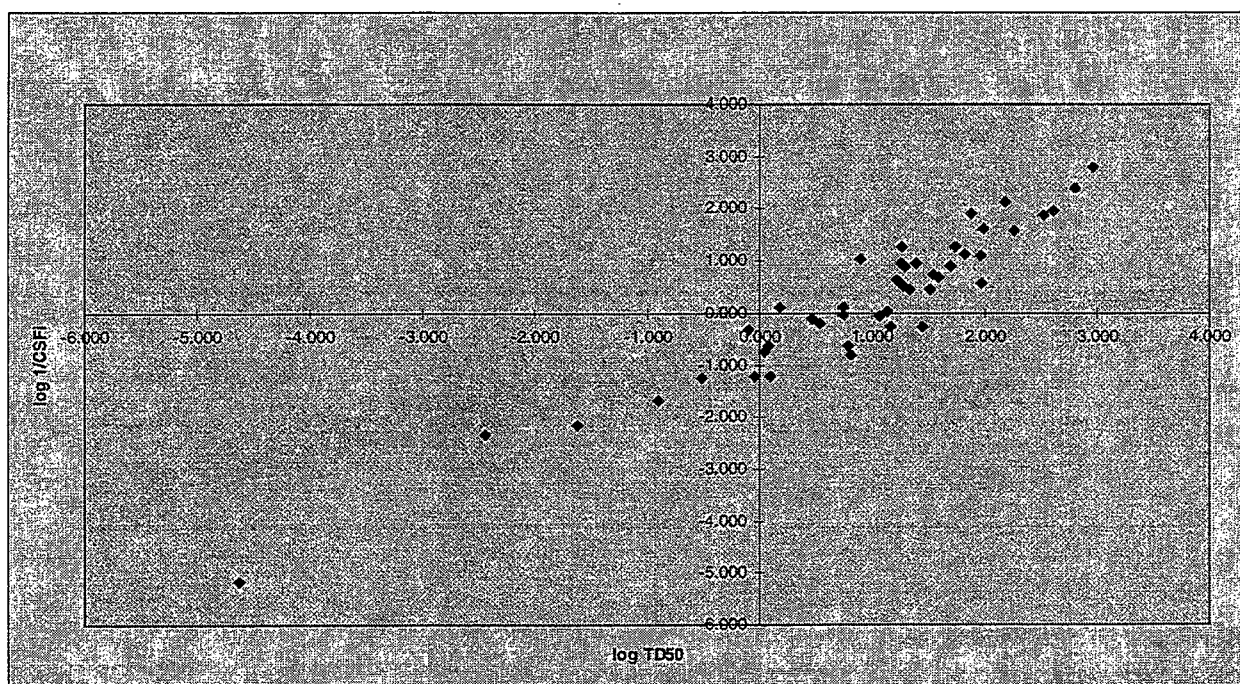


Figure E-1. Correlation of Oral and Inhalation Cancer Slope Factors.

Table E.2. Correlation of TD₅₀s Reported in the Cancer Potency Database and Inhalation Cancer Slope Factors Reported in IRIS and HEAST.

Chemical	CAS #	TD ₅₀ ^a Rat	TD ₅₀ ^a Mouse	TD ₅₀ Geo Mean ^b	Inh CSF ^c	CSF test species ^d	1/CSF	log TD ₅₀ ^e (X)	log 1/CSF (Y)
Carbon tetrachloride	56-23-5	2.29	150	18.53	0.053	b	18.87	1.268	1.276
DDT	50-29-3	84.7	12.3	32.28	0.34	b	2.94	1.509	0.469
Aldrin	309-00-2	-	1.27	*	17	m	0.059	0.104	-1.230
Bis(2-chloroethyl)ether	111-44-4	-	11.7	*	1.16	m	0.86	1.068	-0.064
1,3-Butadiene	106-99-0	261	13.9	*	0.98	m	1.020	1.143	0.009
Chlordane	57-74-9	-	2.99	*	1.3	m	0.77	0.476	-0.114
Chlorobenzilate	510-15-6	-	93.9	*	0.27	m	3.70	1.973	0.569
Chloroform	67-66-3	262	90.3	*	0.08	m	12.50	1.956	1.097
1,1-Dichloroethylene	75-35-4	-	34.6	*	0.175	m	5.71	1.539	0.757
1,3-Dichloropropene	542-75-6	94	49.6	*	0.13	m	7.692	1.695	0.886
Dieldrin	60-57-1	-	0.912	*	16	m	0.0625	-0.040	-1.204
HCH alpha	319-84-6	11.2	6.62	*	6.3	m	0.159	0.821	-0.799
HCH beta	319-85-7	-	27.8	*	1.8	m	0.556	1.444	-0.255
HCH tech.	608-73-1	-	14.8	*	1.8	m	0.556	1.170	-0.255
Heptachlor	76-44-8	-	1.21	*	4.5	m	0.222	0.083	-0.653
Hexachloroethane	67-72-1	55.4	338	*	0.014	m	71.429	2.529	1.854
Methylene chloride	75-09-2	724	918	*	0.0016	m	625.000	2.963	2.796
N-Nitrosodi-n-butylamine	924-16-3	0.691	1.09	*	5.6	m	0.179	0.037	-0.748
1,1,1,2-Tetrachloroethane	630-20-6	-	182	*	0.026	m	38.4615	2.260	1.585
1,1,2,2-Tetrachloroethane	79-34-5	-	38.3	*	0.2	m	5	1.583	0.699
Toxaphene	8001-35-2	-	5.57	*	1.1	m	0.909	0.746	-0.041
1,1,2-Trichloroethane	79-00-5	-	55	*	0.056	m	17.857	1.740	1.252
Acetaldehyde	75-07-0	153	-	*	0.0077	r	129.870	2.185	2.114
Acrylamide	79-06-1	6.15	-	*	4.5	r	0.222	0.789	-0.653
Acrylonitrile	107-13-1	16.9	-	*	0.24	r	4.167	1.228	0.620
Aramite	140-57-8	96.7	158	*	0.025	r	40	1.985	1.602
Azobenzene	103-33-3	24.1	-	*	0.11	r	9.09	1.382	0.959
Bis(Chloromethyl)ether	542-88-1	0.004	0.182	*	217	r	0.00	-2.447	-2.336
Bromoethene	593-60-2	18.5	-	*	0.11	r	9.091	1.267	0.959
Bromoform	75-25-2	648	-	*	0.004	r	250.00	2.812	2.398
1,2-Dibromoethane	106-93-4	1.52	7.45	*	0.77	r	1.299	0.182	0.114
1,2-Dichloroethane	107-06-2	8.04	101	*	0.091	r	10.99	0.905	1.041
1,2-Diphenylhydrazine	122-66-7	5.59	26	*	0.77	r	1.298701	0.747	0.114
Ethylene oxide	75-21-8	21.3	63.7	*	0.35	r	2.857	1.328	0.456
Hexachlorobenzene	118-74-1	3.51	65.1	*	1.6	r	0.625	0.545	-0.204
Hexachlorobutadiene	87-68-3	65.8	-	*	0.077	r	12.987	1.818	1.114
Hydrazine	302-01-2	0.309	2.93	*	17.1	r	0.058	-0.510	-1.233
4,4'-Methylenebis(2-chloroaniline)	101-14-4	19.3	-	*	0.13	r	7.692	1.286	0.886
N-Nitrosodiethylamine	55-18-5	0.024	-	*	151	r	0.007	-1.625	-2.179
N-Nitrosodimethylamine	62-75-9	0.124	0.189	*	49	r	0.020	-0.907	-1.690
N-Nitrosopyrrolidine	930-55-2	0.799	0.679	*	2.13	r	0.469	-0.097	-0.328
Propylene oxide	75-56-9	74.4	912	*	0.013	r	76.9231	1.872	1.886
2,3,7,8-TCDD	1746-01-6	2E-05	2E-04	*	150000	r	0.000	-4.629	-5.176
2,4,6-Trichlorophenol	88-06-2	405	1070	*	0.011	r	90.909	2.607	1.959
Vinyl chloride	75-01-4	19.1	20.9	*	0.3	r	3.333	1.281	0.523
								n	45
								r ²	0.949
								slope	0.983
								intercept	-0.679

a Gold and Zeiger, 1997

b Geometric mean of the TD₅₀ reported for mice and rats (only used when the CSF was derived from both species).

c IRIS, 1998 or HEAST, 1997.

d Test species reported as the basis for the CSF derivation ("b" is both rats and mice, "m" is mice, and "r" is rats).

e Selected to correspond with the CSF test species.

n Number of chemicals with a TD₅₀ and inhalation CSF.

r² Correlation coefficient.

- No data available.

* Not calculated because the CSF was based on a single species.

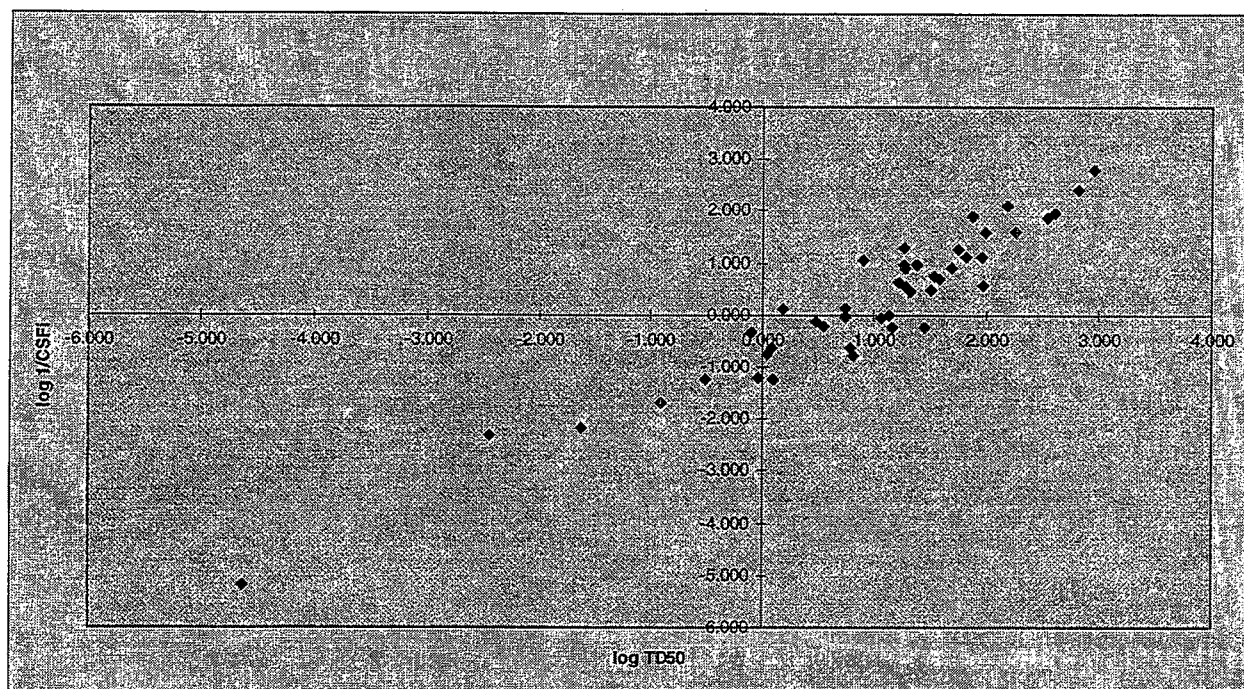


Figure E-2. Correlation of TD_{50} and inhalation cancer slope factors.

